PURE TONE AUDIOMETRIC ASSESSMENT OF HEARING IMPAIRMENT IN PROFESSIONAL DRIVERS

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BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled **"PURE TONE AUDIOMETRIC ASSESSMENT OF HEARING IMPAIRMENT IN PROFESSIONAL DRIVERS"** is a bonafide record work done by **Dr.R.SHANTHIMALAR,** under my direct supervision and guidance, submitted to The Tamilnadu Dr. M.G.R. Medical University in partial fulfillment of University regulation for M.D Branch -V - (Physiology).

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DECLARATION

I **Dr.R.SHANTHIMALAR,** solemnly declare that the dissertation titled **"PURE TONE AUDIOMETRIC ASSESSMENT OF HEARING IMPAIRMENT IN PROFESSIONAL DRIVERS"** has been prepared by me. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, diploma to any other University board either in India or abroad.

This is submitted to The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the rules and regulation for the award of M.D degree Branch – V (Physiology) to be held in April 2012.

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CONTENTS

PURE TONE AUDIOMETRIC ASSESSMENT OF HEARING IMPAIRMENT IN PROFESSIONAL DRIVERS

ABSTRACT:

Objective: To find the prevalence rate of noise induced hearing loss, the hearing threshold levels for high and mid frequency sounds in Professional drivers and to compare both with that of the Office workers.

Design: Cross sectional and descriptive study.

Materials and Methods: This is a cross sectional, descriptive study which was performed on Professional drivers working in Tamilnadu State Transport Corporation with minimum 8 years of driving , 8 hours of exposure to noise and compared with Office workers working for 8 hours per day. The prevalence rate was calculated by observing the characteristic notch at 4 kHz in Pure tone Audiometer. The hearing thresholds were recorded for high and mid frequencies using Pure tone Audiometer.

Results: Significant difference between the two groups was noted ($p<0.001$). The prevalence rate of noise induced hearing loss was higher in Professional drivers (64%) when compared to Office workers (8%). The mean (SD) hearing threshold level for high and mid frequencies was significantly higher in Professional drivers when compared to office workers with $p<0.001$.

Conclusion: Professional drivers have statistically significant higher prevalence rate of noise induced hearing loss and statistically significant higher hearing thresholds compared to Office workers.

Key Words: Pure tone Audiometer, Hearing threshold, Notch at 4 kHz, Professional drivers, Office workers.

INTRODUCTION

Noise is one of the most pervasive problems in today's occupational environment affecting workers in various professions. Traffic noise is a major source of environmental pollution in developed and developing nations. Many field surveys have been conducted evaluating the traffic noise environment in several countries including India. **Kumar & Jain 1994, Chakraborthy *et al*., 1997, Mukherjee *et al*., 2003, Hydel *et al*., 2006, Zannin 2008**.

Occupational noise induced hearing loss refers to a hearing loss caused by loud sounds experienced in a work place. The hearing loss is caused by exposure to loud sounds at 85 dB or over, through a prolonged period of time **Rabinowit, 2000.**

The National Institute on Deafness and other Communication Disorders **(NIDCD),** National Institute of Health **(NIH),** National Institute on Environmental Health Sciences **(NIEHS**) and the National Institute for Occupational Safety and Health **(NIOSH)** have noted that because of noise in our society, hearing loss is occurring much earlier in life than would have been expected just 30 years ago.

Estimates from National Institute of Health, **Morata, 2007** suggest that one third of hearing losses are caused due to noise

exposure. **Henderson, 2006** estimated that 35-51% have hearing loss due to noise trauma when looking at occupational noise exposed populations. It has also emerged that up to 10% of younger people are more at risk of developing noise induced hearing loss Borchgrevink**, 2003, Hodgetts, *et al.*, 2009.**

Konig S A, Van Laer L, and Van Camp G, 2009 stated that approximately 30 million American workers are exposed daily to hazardous noise levels at work and 20 % of European workers are exposed to injurious noise levels half their work time.

Approximately 0.5 million population of Indian metropolitan cities are exposed to noise levels that are potentially hazardous to their hearing. The gradual progression of hearing loss due to noise is a significant and permanent handicap for the affected individual **Frank's *et al,* 1996**. Noise induced hearing loss is most likely to occur in an occupational setting where sounds are either presented in a continuous manner or impulsively **Axelsson, 1979**. Hearing handicap is usually denoted as an average hearing threshold level of greater than 25 dB (A) for both ears at selected frequencies **Prince *et al*., 1997.**

The source of most outdoor noise worldwide is mainly transportation systems including motor vehicle noise, air craft noise

and rail noise. Noise pollution in urban cities is steadily increasing over the years. Proportion of people exposed to noise is greatly increasing. Of those, drivers are at high risk to be affected by traffic noise and other urban noise **Janssen *et al*., 2001. Zannin 2008,** measured traffic noise levels in urban buses and concluded that bus engine is the main source of noise. **Anon 2004** assessed the road traffic noise in Kolkata city of India and it ranged between **66.9 – 96.9 dB (A).** The majority of drivers remain unaware of the health effects of noise on their hearing ability as this is an insidious process and takes long time to become overt. Adverse health effects due to noise include both auditory as well as non auditory Kuzli **et al., 2000, Pope Bogoch *et al*., 2005**, and **Dockery, 2006.**

Noise can hamper performance of daily tasks, increase fatigue and cause irritability. It can reduce efficiency in performing daily tasks by reducing attention to tasks. Noise also makes speech communication harder. More concentration of energy is needed to speak louder above the noise. It is a physical strain to carry on even an enjoyable conversation in the presence of noise.

Thus noise is both a public health hazard and an environmental pollutant. We need to eliminate unwanted noise when we can. When noise cannot be eliminated, we should keep it as low as possible.

Hence this study has been taken up to study the effect of years of noise exposure on hearing in Professional drivers and also to compare it with Office workers. It is hoped that this study will contribute to the knowledge of the effects of noise pollution on hearing and will improve the public awareness of the hazardous effects of noise.

AIM & OBJECTIVES OF THE STUDY

1. To find the prevalence of hearing impairments due to noise exposure in Professional drivers and to compare it with Office workers using Pure tone Audiometer.
2. To compare the prevalence of 4 kHz notch between the Professional drivers and Office workers.
3. To compare the mid and high frequency hearing thresholds of both ears among drivers with 4 kHz notch.
4. To compare the mid and high frequency hearing thresholds of both ears among the professional drivers.
5. To find the correlation between “Period of noise exposure” and “Hearing loss”.
6. To find out the ear advantage between right and left ears.

REVIEW OF LITERATURE

NOISE

Historical Aspects of Sound

In the 6th century B.C **Pythagoras** of **Samos** observed someone playing a stringed instrument and as he observed the string being plucked, he related the amplitude of its vibration with the perceived loudness of the sound. He also noted that when the vibration stopped altogether, the sound stopped as well. He even saw that the shorter strings vibrated more rapidly, and that this more rapid vibration seemed to produce a shriller, higher pitched sound.

By 400 B.C a member of the Pythagorean school, **Archytus** of Tarentum postulated that sound was produced by the striking together of objects. From this he also gathered that a fast motion resulted in a high pitch and slow motion resulted in a low pitch.

Around 350 B.C **Aristotle** observed that the vibrating string was actually striking the air. He also concluded that each bit of air struck a neighboring bit of air, which in turn struck another bit, and so on. From this Aristotle hypothesized that air was needed as a medium through which sound could be conducted.

The invention of the tuning fork in 1711, by **John Shore** and its further development by Frenchman **Rudolph Koning** eased the study of sound considerably. Later breakthroughs in sound were made when in 1842, **Christian Doppler** first identified and quantified the change in pitch that occurred when a source of sound moves toward or away from a stationary observer, or an observer moves toward or away from a stationary source of sound. This effect now bears his name and is known as the **Doppler Effect**.

Other modern contributors to the study of sound included **Helmholtz**, **Lord Rayleigh, Weber, Fechner, Fletcher, Bekesy and Mach**, who observed the Mach cone and whose name gives us the **Mach number**, which is "how fast an object is moving compared to the speed of sound".

An expert on noise**, K.D.Kryter, 1996** in his text Handbook of Hearing and the effects of noise defined noise as "Acoustic signals which can negatively affect the physiological or psychological well being of an individual." The combined disciplines Psychology and Acoustics define noise as an unwanted sound.

Relation between Sound, Loudness and Noise:

Sound, Loudness and Noise are interrelated. The vibrating objects cause alternating phases of compression and rarefaction which spread out as sound waves and are perceived by the auditory mechanism as Sound.

Sound that travels through air has a frequency at which the waves vibrate and the intensity of each vibration produces the sense of Loudness.

If a sound contains many components which have frequencies that are not simple multiples of the fundamental, it is referred to as Noise.

All **noisy sounds** are of high amplitude and low frequency with completely **irregular wave pattern**. The amplitude of sound waves determines the loudness, while frequency of sound waves determines the pitch of the sound. The greater the amplitude, the louder the sound. The greater the frequency, the higher the pitch.

Measurement of Sound:

Loudness is measured by a decibel scale (dB). dB scale is a log scale, so a value of zero dB does not mean the absence of sound but a sound level of intensity equal to that of the reference.

Zero decibel = reference sound. The standard sound reference level adopted by the **Acoustical society of America** corresponds to **'0' dB** at a pressure level of **0.0002 dynes/cm^2,** a value that is just at the auditory threshold for the average human.

The sound frequencies audible to humans range from **20-20,000 cycles/sec.** The threshold of the human ear varies with the pitch of the sound, the greatest sensitivity being in the **1000-4000 Hz** range. The number of pitches that can be distinguished by an average individual is about **2000.** Pitch discrimination is best between **1000-3000 Hz.**

The human audible sound intensity range is **0-120 decibels**. To mimic the response of the human ear and to allow for the variation in ear sensitivity to different frequencies, noise meters apply a weighting to the sound intensities and express the readings as dB(A) i.e. decibels weighted by the 'A' scale as defined by International standards. Its effect is to progressively reduce the sensitivity of the sound level meter with decreasing frequency below 1 kHz. 'A' weighted sound pressure level is used as an approximation to the sound level at the threshold of hearing in a free fluid and this 'A' weighted measurement is often preferred in the calculation of noise exposure. This weighting should be assumed unless otherwise stated.

Noise levels are measured in decibels (dB). The higher the decibel level, the louder the noise. Sounds louder than 80 decibels are considered to be potentially hazardous. According to **WHO,** minimum limit of noise exposure for those working in noisy environment is 85 dB (A) for 8 hours per day for 5 days a week. Moderately high noise exposure is 85-90 dB (A) and high noise exposure is more than 90 dB (A).

According to **ISO** standard, noise levels on roads **should not exceed 70 dB** but the fact is otherwise. Noise levels on roads average around 90 dB (A). Continuous exposure to sounds over 85 dB (A) may cause permanent hearing loss. Both the amount of noise and the length of exposure determine the amount of damage. Noise induced hearing loss is usually gradual and painless, but unfortunately permanent.

The damage caused by noise trauma depends on several factors:

1. Frequency of noise: A frequency of 2-3 kHz causes more damage than lower or higher frequencies.
2. Intensity and duration of noise: As the intensity increases, the damage produced will be more.
3. Continuous sound is more harmful than interrupted sound.

The noise chart gives the average decibel levels for everyday sounds around us.

120-160 dB	=	Painful. (Jet plane take off, rock music peak).
90-110 dB	=	Extremely high. (Traffic, Subway).
60-80 dB	=	Very loud. (Busy street, Alarm clock, Vacuum Cleaner).
40-50 dB	=	Moderate (Quiet room, moderate rainfall).
30 dB	=	Faint. (Whisper, Quiet library).

Equal energy principle states that similar damage to cochlea will occur from loud sounds of a short duration as to soft sounds of a longer duration. **Konings, van Laer and Van camp** noted noise is harmful to human ears when exposed to 85 dB(A) for 8 hours and increasing noise intensity by 3 dB doubles the energy output thus making the noise twice stronger than before. Therefore exposure time must be reduced by half, for every additional 3 dB of sound to maintain safe noise exposure limit. Noise at **Work Regulations, 2005** suggests, personal hearing protection to be provided where noise levels reach 85dB (A) or more.

Permissible limit of sound exposure & duration

National Institute for Occupational safety and Health recommended the permissible level of sound exposure and the duration:

Level &Exposure	Permissible exposure duration
85 dB	8 hours
88 dB	4 hours
91 dB	2 hours
94 dB	1 hour
97 dB	30 minutes
100 dB	15 minutes
110 dB	1 minute

PURE TONE AUDIOMETRY

Introduction

Pure tone audiometry is a science for ascertaining the hearing threshold level of a subject for pure tone sounds of various frequencies. The result when plotted graphically is called pure tone audiogram. The instrument used for this is an electronic device called Pure tone Audiometer.

Audiometric testing is the accepted standard test for measuring hearing loss. Because pure tone audiometric results have significant influence on the medical, legal, educational, occupational and social outcomes it is critical that procedures be standardized and consistent. These guidelines represent a consensus of recommendations found in standards such as Methods for Manual pure tone Threshold Audiometry (**ANSI S3-21-2004) American National Standards Institute, 2004** with particular emphasis on the suggestions of **Reger 1950 , Carhart and Jerge 1959.**

Parts & Calibration:

Pure tone audiometer consists of a pure tone generator, an amplifier, and an attenuator. The generator generates pure tone sounds of various frequencies of 125, 250, 500, 1000, 1500, 2000, 3000, 4000, 6000, 8000 & 10,000 Hz. Each tone can be separately amplified by the amplifier to a maximum of 100 or 110 dB in most frequencies except the very low and high frequencies. The tones are attenuated by an attenuator dial.

The Audiometer is connected to standard earphones or to a bone conduction vibrator through which the sound is presented to the subject's ears. The Audiometer is operated by means of a noiseless switch called interrupter which will introduce or interrupt a tone.

Calibration of the instrument has to be done perfectly. An electronic calibration has to be done once in 6 months but each day before starting the test, biological calibration should be done.

Thus Pure tone Audiometry gives an idea about the type of hearing loss and it gives a measure of the degree of hearing loss. If this test is done routinely as a screening test for those who are exposed to noise, hearing loss can be diagnosed at an earlier date and preventive and protective measures can be followed.

SENSE OF HEARING

Physiological Anatomy of the Ear

The organ of hearing consists of an outer, middle, and inner ear. The pinna helps to locate the sound. The external auditory canal channels sound pressure to the tympanic membrane. The tympanic membrane divides the outer from the middle ear. The middle ear cavity normally air filled, is connected to the throat by a narrow passage the Eustachian tube, through which air can escape from the middle ear cavity more easily than it opens to air.

The three middle ear ossicles are the malleus (hammer), incus (anvil), and stapes (stirrup) articulate with one another from tympanic membrane to oval window. When the tympanic membrane is vibrated, the ossicles are set into motion whereby the stapes transmits the vibrations to the oval window and fluid filled scala vestibule.

There are two middle ear muscles the tensor tympani, which is attached to the handle of malleus and stapedius which is attached to the stapes. Sound delivered to one ear elicits contralateral as well as ipsilateral reflex contraction of the middle ear muscles. Middle ear muscles provide reflex protection of the cochlea from damaging loud sounds.

Physiological Anatomy of the Inner Ear

Development of the inner ear starts in the third week and is completed by the 16th week of intra uterine life. Inner ear is well protected and lies inside the petrous canal.

The membranous portion of the inner ear originates from an ectodermal thickening adjacent to the hindbrain called “Otic placode”. In humans otic placode is evident at the 3rd week of embryonic development. The cochlea begins as an evagination from the ventral portion of the otocyst starting at around the 5th week of gestation. After extending ventrally, the cochlear duct begins coiling such that it has formed $1^1/_2$ turns by 8 th week, 2 turns by 10 th week, and has completed the normal $2^1/_2$ turns by 25 weeks of gestation. The organ of corti develops from the posterior wall of the cochlear duct.

Inner Ear

This is a compartment filled with fluid and housed in a system of long cavities and tunnels called the osseous labyrinth inside which is the membranous labyrinth. It consists of the sense organs of hearing and equilibrium which open into a cavity known as the vestibule. Within the vestibule, are two membranous sacs the saccule and utricle which communicate with each other.

Cochlea: It resembles a common snail. It forms the anterior portion of the bony labyrinth. It is 5 mm from base to apex, 9 mm around its base; length of the tube is 30 mm. Bony cochlea is a coiled tube making 2.5 to 2.75 turns round a central pyramid of bone called the modiolus. Around the modiolus winding spirally like the thread of a screw, is a thin plate of bone called osseous spiral lamina which gives attachment to the basilar membrane. The bony cochlea contains three compartments (a) Scala vestibule (b) Scala tympani and (c) Scala media.

The cochlea, including the stria vascularis receives important autonomic innervations. Autonomic sympathetic fibers release norepinephrine to control the vascular supply. The difference in chemical composition between endolymph and perilymph creates a steady potential across the basilar membrane, approximately double that across ordinary cell membranes **Tasaki, 1954**.

The scala vestibule and scala tympani are filled with perilymph and communicate with each other at the apex of the cochlea through an opening called helicotrema. The scala vestibule originates at the oval window and scala tympani ends at the round window.

The scala media is filled with endolymph and is rich in potassium with a voltage of +80 mv relative to the perilymph. The endocochlear potential, the highest transepithelial voltage in the body is the main secretary force for sensory transduction in both inner and outer hair cells. A highly vascularised tissue stria vascularis secretes K^+ into the scala media and the K^+ gradient between endolymph and perilymph generates the endocochlear potential. Loss of this endocochlear potential is a frequent cause of hearing loss.

Cochlear duct (Scala media or membranous cochlea)

It appears triangular and its three walls are formed by:

1. Basilar membrane which supports the organ of corti
2. Reissner's membrane which separates scala media from scala vestibule and
3. Stria vascularis which contains vascular epithelium and is concerned with secretion of endolymph.

The length of the basilar membrane increases from the basal coil to the apical coil increasing from a length of about 0.04 mm near the oval and round windows to 0.5 mm at the tip of the cochlea, a 12 fold increase in length. The diameter of the fibers, decrease from the oval window to apex, so their overall stiffness decreases more than 100

fold. Thus the stiff short fibers near the oval window of the cochlea vibrate best at a very high frequency, and the long limber fibers near the tip of the cochlea vibrate best at a low frequency. On the basilar membrane or base of the triangle there are supporting cells and specialized cochlear hair cells containing auditory receptors. These cells form the spiral organ of corti, the sensory organ that responds to vibration by initiating nerve impulses that are then perceived as hearing by the brain.

Organ of corti: It is the sense organ of hearing and is situated on the basilar membrane. Components of organ of corti are:

1. Tunnel of corti.
2. Hair cells (inner, outer hair cells)
3. Supporting cells (Deiter's cell, Hensen's cell and Claudius cell).
4. Tectorial Membrane
5. Reticular lamina.

Hair cells: Hair cells are important receptor cells of hearing and transduce sound energy into electrical energy. Hair cells are embedded in an epithelium made up of supporting cells, with the basal end in close contact with afferent neurons. The stereocilia, the processes that

are present in all hair cells have parallel filaments of actin. The actin is coated with various isoforms of myosin. Along an axis towards the kinocilium, which is the nonmotile cilium, the stereocilia increase progressively in height and along the perpendicular axis all the stereocilia are at the same height. The hair cells lie within a matrix of supporting cells, with their apical ends facing the endolymph of scala media. The stereocilia of inner hair cells float freely in the endolymph and that of outer hair cells project into the tectorial membrane which has its attachment only along one edge like a hinge which makes it to tilt up and down freely.

Differences between inner and outer hair cells.

	Inner Hair cells	**Outer Hair cells**
Total	3,500	12,000
Rows	1	3-4
Shape	Flask shaped	Cylindrical
Nerve supply	Primarily afferent, very few efferent	Primarily efferent, very few afferent
Function	Transmit auditory stimuli	Modulate function of inner hair cells
Vulnerability	More resistant	Easily damaged by high intensity sounds

Supporting cells:

The inner hair cells are supported by inner phalangeal cells, while the outer hair cells are supported by Deiter's cells. Hensen's cells that lie outside the outer hair cells support the organ of corti. The cells of Claudius line the lower surface of external spiral sulcus.

Tectorial membrane:

It consists of gelatinous matrix with delicate fibers and it overlies the organ of corti. The vibrations of the perilymph cause movement of tectorial membrane and movement of hair cells which stimulates the cochlear nerve endings. The shearing force between the hair cells and tectorial membrane produces the stimulus to hair cells. The cochlear nerve fibers ramify between the hair cells.

Reticular lamina: It is a tough membrane supported by rods of corti.

Stria vascularis:

It forms the lateral wall of the cochlear duct. It plays an active role in the maintenance of ionic composition and electrical potential of endolymph.

Blood supply:

The entire inner ear receives its arterial supply through labyrinthine artery a branch from anterior inferior cerebellar artery.

Physiology of Hearing

Any vibrating object causes waves of compression and rarefaction and is capable of producing sound. In the air at 20^0 C and at sea level sound travels at a speed of 344 metres/sec. It travels faster in liquids than in the air. When sound energy has to pass from air to liquid medium, most of it is reflected because of the impedance offered by the liquid.

Mechanism of hearing

A sound signal in the environment is collected by the pinna, passes through external auditory canal and strikes the tympanic membrane. Movements of footplate of stapes cause pressure changes in the labyrinthine fluids which move the basilar membrane. This stimulates the hair cells of the organ of corti. It is these hair cells which act as transducers and convert the mechanical energy into electrical impulses which travel along the auditory nerve.

Thus mechanism of hearing can be divided into:

1. Mechanical conduction of sound. (**Conductive apparatus**)
2. Transduction of mechanical energy to electrical impulses. (**Sensory system of cochlea)**
3. Conduction of electrical impulses to the brain. (**Neural pathway)**

Conduction of sound:

Any sound made in the air cannot be heard by a person in water because 99% of sound energy is reflected away from the surface of water due to impedance offered by it. In the ear this type of loss of sound energy is compensated by the middle ear which converts sound of greater amplitude but lesser force to that of lesser amplitude but greater force by its "Impedance matching mechanism".

Impedance matching mechanism includes;

a. **Lever action of the ossicles**: Handle of malleus is 1.3 times longer than long process of the incus, providing a mechanical advantage of 1.3. Movement of the tympanic membrane which is more at the periphery than at the center where malleus is attached also provides some leverage.

b. **Hydraulic action of tympanic membrane**:

The effective vibratory area of tympanic membrane is 55mm^2 which is much larger than the area of stapes footplate which is 3.2mm^2. Hence areal ratio between the two being 17:1 and this is the mechanical advantage provided by the tympanic membrane. This 17 fold difference times the 1.3 fold ratio of the lever system causes about 22

times as much total force to be exerted on the fluid of the cochlea as is exerted by the sound waves against the tympanic membrane.

c. Phase differential between oval and round window:

In normal ear sound pressure waves never reach the oval and round window in the same phase, due to the presence of tympanic membrane, middle ear and air cushions. If the sound waves strike both the windows simultaneously, they would cancel each other's effect with no movement of the perilymph and no hearing. Hence, normally sound waves reach oval window earlier than round window which is an added advantage of hearing.

Transduction of mechanical energy to electrical impulse:

Movements of the footplate of stapes, transmitted to the cochlear fluids, move the basilar membrane setting up shearing force between the tectorial membrane and the hair cells. The distortion of hair cells gives rise to cochlear microphonics which triggers the nerve impulse.

The vibration of the basilar membrane excites the hair endings. Upward movement of the basilar fibers rocks the reticular lamina upward and inward toward the modiolus. When the basilar membrane moves downward, the reticular lamina rocks downward and outward. The inward and outward motion causes the hairs on the hair cells to

shear back and forth against the tectorial membrane. Thus the hair cells are excited whenever the basilar membrane vibrates.

Hair cell Receptor potential and excitation of Auditory nerve fibres:

Each hair cell has about 100 stereocilia on its apical border. They become progressively longer on the side of the hair cell away from the modiolus, and the tops of the shorter stereocilia are attached by thin filaments to the backsides of their adjacent longer stereocilia. Very fine processes called **"Tip links"** tie the tip of each stereocilium to the side of its higher neighbour, and at the junction are cation channels in the higher process that appear to be mechanically sensitive.

When the shorter stereocilia bent toward the higher, the tips of the smaller stereocilia are tugged outward from the surface of the hair cell. This causes a mechanical transduction that opens 200-300 cation conducting channels, allowing rapid movement of positively charged potassium ions from the surrounding scala media fluid into the stereocilia and produce depolarization of the hair cell membrane resulting in the release of the neurotransmitter Glutamate by the hair cells which initiates depolarization of neighbouring afferent neurons.

When the basilar fibers bend towards the scala vestibuli, the hair cells depolarize, and in the opposite direction they hyperpolarize, generating an alternating hair cell receptor potential. This in turn stimulates the cochlear nerve endings that synapse with the bases of the hair cells.

The outer hair cells respond to sound, but depolarization makes them shorten and hyperpolarization makes them lengthen. They do this over a very flexible part of the basilar membrane, and this action increases the amplitude and clarity of sounds. These changes in outer hair cells occur in parallel with changes in **Prestin**, a membrane protein of the outer hair cells.

Action potentials in auditory nerve fibers:

The frequency of the action potentials in the auditory nerve fiber is proportional to the loudness of the sound stimuli. At low sound intensities, each axon discharges to sounds of one frequency, and at higher sound intensities, the individual axons discharge to a wider spectrum of sound frequencies, particularly to frequencies lower than that at which threshold stimulation occurs. The major determinant of the pitch is the place in the organ of corti that is maximally stimulated. The travelling wave set up by a tone produces peak depression of the

basilar membrane, and consequently maximal receptor stimulation, at one point. The distance between this point and the stapes is inversely related to the pitch of the sound, with low tones producing maximal stimulation at the apex of the cochlea and high tones producing maximal stimulation at the base. The frequency of action potentials in an auditory nerve determines the loudness of a sound.

Auditory neural pathway:

The nerve fibers from the spiral ganglion of corti enter the dorsal and ventral cochlear nuclei where the fibers synapse, and second order neurons pass mainly to the opposite side of the brainstem to terminate in the superior olivary nucleus while few fibers pass to the same side. From the superior olivary nucleus, the auditory pathway passes upward through the lateral lemniscus. Few fibers terminate here and many bypass this nucleus and travel on to the inferior colliculus, the center for auditory reflexes, medial geniculate body in the thalamus, auditory radiation, and to the auditory cortex **Broadmann's area 41**, located in the **superior gyrus of temporal lobe**. High degree of spatial orientation is maintained in the fiber tracts from the cochlea all the way to the cortex. High frequency sounds are located posteromedially, and low frequency sounds are located anterolaterally in the auditory cortex.

In the primary auditory cortex, most neurons respond to inputs from both ears, but strips of cells are stimulated by input from the contralateral ear and inhibited by input from the ipsilateral ear.

The olivocochlear bundle is a prominent bundle of efferent fibers in each auditory nerve that arises from both ipsilateral and contralateral superior olivary complexes and ends primarily around the bases of the outer hair cells of the organ of corti.

The responses of individual second order neurons in the cochlear nuclei to sound stimuli are like those of the individual auditory nerve fibers. The frequency at which sounds of the lowest intensity evoke a response varies from unit to unit. With increased sound intensities, the band of frequencies to which a response occurs becomes wider.

The major difference between the responses of the first and second order neurons is the presence of a sharp "cutoff" on the low frequency side in the medullary neurons. This greater specificity of the second order neurons is probably due to an inhibitory process in the brain stem.

Other cortical areas concerned with audition:

Broadmann's area 22 is concerned with the processing of auditory signals related to speech. During language processing, it is

much more active on the left side than on the right side. Area 22 on the right side is more concerned with melody, pitch and sound intensity. The auditory pathways are very plastic and are modified by experience. Musicians have additional cortical plasticity. In these persons the size of the auditory areas activated by musical notes is increased. A portion of the posterior superior temporal gyrus known as **planum temporale** is involved in language related auditory processing and is larger than normal on the left side in musicians and others who have perfect pitch.

A functional difference between the two hemispheres is apparent with dichotic listening. The right ear (left hemisphere) reveals a better score for verbal tests, while the left ear (right hemisphere) tests better for recognition of music.

Theories of Hearing:

Many theories are postulated to explain the mechanism by which the pitch of the sound is appreciated or the frequency is analysed.

1. **Telephone or Frequency theory** by **Rutherford**, **1886** states that the basilar membrane vibrates as a whole, aperiodically at the same frequency as the sound wave and sets up nerve impulses of the same frequency. He also stressed that sound analysis is a function of cerebral cortex and not that of the cochlea.

2. **Volley theory** states that impulses are discharged over separate fibres as a scattered volley, and not synchronously, so that a group of fibres may discharge as very high frequencies.
3. **Resonance theory** by **Helmholtz, 1863** states that a series of resonators are in the cochlea, each responding to a different frequency.
4. **Place theory:** According to this, the entire cochlea is a tuned structure with different parts of the basilar membrane responding to different frequencies, with the basal part responding to higher frequencies and the apical part responding to lower frequencies. Intermediate frequencies evoke responses in between base and apex in the descending order of frequency.
5. **Travelling wave theory:** The movements of the footplate of the stapes set up a series of travelling waves in the perilymph of the scala vestibuli. When the sound wave enters the oval window it causes the bending of the basilar membrane in the direction of the round window. The elastic tension that has been built up in the basilar fibers initiates a fluid wave that travels along the basilar membrane toward the helicotrema. Each wave is weak at the outset, but becomes strong when it reaches that portion of the basilar membrane that has a natural resonant frequency equal to the respective sound frequency. Thus, a

high frequency sound wave travels only a short distance along the basilar membrane and dies, a medium frequency sound wave travels about half way and dies, and a very low frequency sound wave travels the entire distance along the membrane.

EFFECTS OF NOISE ON HEARING

Historical aspects of noise induced hearing loss

A causal connection between loud noises and hearing loss has no doubt been recognized thousands of years ago. The earliest extent reference to the effect of noise on hearing appears to be an observation recorded in the first century A.D, by **Pliny the Elder** in his Natural History, when he noted that persons living near the cataracts of the Nile were "Strucken deaf" **Bacon, 1627**. It is no wonder, then that the problem of noise induced hearing loss and its prevention are assuming greater and greater importance all over the world and it can be prevented or at least minimized if reasonable precautions against noise exposure are taken.

The deafness of artillery men was well known in the Napoleonic wars and hearing loss was noted among industrial workers since 1800.

Barr of Glasgow produced an excellent epidemiological study of 'Boiler Makers'. Deafness among shipyard workers in 1880 described rubber ear plugs to protect hearing.

Haberman described the histology of the cochlea in a deaf railway worker and attributed it to noise. Thus the causes, pathology and epidemiology of noise induced hearing loss were already known

and techniques of hearing conservation were being applied by the beginning of the 12 th century.

Pathophysiology of noise induced hearing loss

High levels of noise have a significant impact on the auditory system and overall physiology of humans **Kryter 1985.**

Exposure to impulse noise causes anatomical changes that range from distorted stereocilia of the inner and outer hair cells to complete absence of the organ of corti and rupture of Reissner's membrane. After a few minutes of exposure to impulse noise, edema of the striavascularis appears and persists for several days.

Cochlear inflammatory response is initiated in response to acoustic trauma and involves the recruitment of circulating leucocytes to the inner ear. Exposure to sufficiently intense noise for a long enough duration results in damage to the inner ear and thus decreases one's ability to hear, decreases the quality and clarity of auditory perception. **Sataloff, 1965,** describes the effect **"Sound induced motion"** of the fluid in the cochlea induces shearing and bending movements of the hair cells in the organ of corti, which in turn result in electrical stimuli transmitted to the auditory nerve. The damage continues in the most vulnerable elements of the ear the sensory cells

themselves, in particular their stereocilia and the rootlets which anchor them to the circular plate **Liberman 1990**.

Changes in hair cells

Outer hair cells are more susceptible to noise exposure than inner hair cells. Exposure to moderate intensity noise for several minutes or hours initially results in a **Temporary Threshold Shift** only. This Temporary threshold shift is anatomically correlated with decreased stiffness of the stereocilia of the outer hair cells leading to floppy cilia. The stereocilia become disarrayed and at this stage they respond poorly. Temporary threshold shift reflects reversible buckling of the pillar cell bodies **Nordmann *et.al*., 2000**, Temporary strial edema and reduction of the endocochlear potential **Hirose and Liberman, 2003,** or Excitotoxic damage to afferent fibers **Pujol and Puel, 1999**. In this Temporary threshold shift the auditory thresholds eventually returns to its original level.

Permanent Threshold Shift occurs after repeated noise exposure which causes an irreversible increase in the hearing threshold **Gelfend 2009**. Permanent threshold shift is associated with fusion of adjacent stereocilia **Mathur &Roland 2009**, loss of stereocilia and death of hair cells. Loss of stereocilia is due to the formation of oxygen

and nitrogen free radicals leading to antioxidant mechanisms being inundated in the hair cells when they respond to intense noise **Lynch & Kil, 2005.** This combination causes metabolic exhaustion leading to cell death. Permanent noise damage initially consists of degeneration of hair cells. Although both types of hair cells may degenerate, outer hair cells are more sensitive to noise than inner hair cells. With long exposures or a more intense noise, there is further loss of outer hair cells, inner hair cells and supporting cells which are the outer and inner pillars. If the cell loss is confined to a narrow region of the organ of corti, a "focal" hair cell lesion develops. **Bohne and Clarke** define a focal lesion as a region in which 50% or more of the outer hair cell and /or inner hair cells are missing over a distance of at least 0.03 mm. **Johnsson and Hawkins, 1976** have termed focal hair-cell lesions as **'Cookie-bite'** defects. With severe exposure, injury can proceed from a loss of adjacent supporting cells to complete degeneration of afferent fibers in the organ of corti **Slepecky, 1986; Saunders *et al*., 1991.** A lesion in which no recognizable cells of the organ of corti remain on the basilar membrane is termed as **'OC wipeout'** by **Bohne and Clark**.

Changes in Organ of Corti:

Noise damage begins as scattered losses of hair cells. With short duration, moderate level exposure, the damage does not appear to spread to adjacent cells.

In the reticular lamina, degenerated hair cells are replaced by phalangeal scars which are formed by enlarged processes from outer pillar cells, Deiter's cells or inner phalangeal cells. When the hair cells initially degenerate, defects or holes are left in the reticular lamina for a period of time before the phalangeal scars form. These holes provide a route for endolymph to enter the fluid spaces of the organ of corti **Bohne, 1976, Rabbitt, 1983**. When most of the outer hair cells degenerate in a focal region the intermixing of endolymph and perilymph produces secondary loss of adjacent supporting cells, sensory cells, and nerve fibers until an entire region of organ of corti has been lost. Within Organ of corti wipeouts, the endolymph boundary consists of a single layer of squamous epithelium which replaces the Organ of Corti, Claudius, Hensen's and Deiter's cells on the basilar membrane and seals the open ends of the tunnel and Nuel spaces.

Once inner hair cell loss reaches moderate proportions, there is a beginning of loss of myelinated nerve fibers, the peripheral processes of the spiral ganglion within the osseous spiral lamina **Bohne *et al*., 1987.** Eventually, the spiral ganglion cells which originally innervated the degenerated portion of the organ of corti are progressively lost, including their central processes which form the auditory portion of the eighth nerve.

Once degeneration of the spiral ganglion cells has begun, there is a corresponding degeneration within the central nervous system including the cochlear nuclei, superior olive and inferior colliculus **Kim *et al*., 1997; Morest *et al*., 1998**.

Examination of noise damaged human temporal bones has shown that lesions in the high frequency region consist of near total loss of the Organ of Corti, its afferent innervations **Bredberg, 1968, Johnson and Hawkins, 1976,** and complete destruction of organ of corti **Gelfand 2009**.

Causes for degeneration of organ of corti

1. Reduced blood flow during noise exposure **Hawkins, 1971** leading to hypoxia and the release of reactive oxygen species in the cochlea. **Quirk *et al*., 1992**. Reduced circulation in the

cochlea is mediated by circulating vasoactive peptide angiotensin **Wayne S. *et al.***

2. Metabolic exhaustion of the stimulated sensory cells results in cell death **Lim and Dunn, 1973**.
3. Excessive release of neurotransmitter during the exposure leading to excitotoxic damage of afferent nerve fibers and terminals **Pujol, 1992**.
4. Intermixing of cochlear fluids through the damaged reticular lamina **Bohne and Rabbitt, 1983**.

Animal studies show:

1. Involvement of Src-PTK (protein tyrosine kinase) signaling cascade in both the metabolic and mechanically induced irritation of apoptosis in the sensory cells of the cochlea.
2. Decreased endolymphatic O_2 tension directly related to the duration of intensity of noise exposure.
3. Involvement of glucocorticoid signaling pathways in the cochlea.
4. Blood stagnation in strial capillaries leading to strial dysfunction **Yamane H, Nakai Y *et al.*, 1995**.

Molecular mechanisms involved in Noise Exposure

Calcium homeostasis in hair cells and spiral ganglion neurons is maintained by regulatory proteins such as calmodulin and calbindin **Hansen *et al*., 2003; Hackney *et al*.,2005**, and by several types of calcium channels **Parks, 2000; Lopez *et al*., 2003**. In the noise exposed cochlea, calcium may participate in both hair cell and neuronal damage **Minami *et al*., 2004**. So dysregulation of calcium homeostasis results in noise induced hearing loss and Calcium channel antagonists promote hair cell survival. **LeFebvre et al., 2002; LePrell et al., 2006, Henderson, 2006.**

Genetic factors related to noise induced hearing loss:

1. Potassium recycling genes **Van Laer *et al.,* 2005**.
2. Heat shock proteins **Yuan *et al.,* 2005**, **Yang *et al*., 2006,**

Biological factors related to Noise induced hearing loss:

Melanization is protective. Fair haired, blue eyed people are more sensitive to the harmful effect of intense sound than brown haired, brown eyed people **Hood *et al.,* 1976.**

Women have slightly "tougher" ears than men. Noise induced hearing loss affects males more than females **Rabinowitz, 2000**.

Short stature **Barrenas et al., 2005,** and Young people are at risk of developing NIHL **Borchgrevink, 2003**, **Hodgetts, *et.al.,* 2009**.

Race may be an influential factor with Caucasians, more sensitive than Africans **Morata, 2007**.

Noise induced hearing loss in humans commonly begins in the high frequencies **Rabinowitz, 2006** around 4 kHz, regardless of the frequency content of the noise. With continued exposure, the threshold at 4 kHz worsens and the hearing loss spreads to involve higher frequencies **Schmuzigert *et al.,* 2006, Tambs *et al.,* 2006** and lower frequencies **Taylor *et al*., 1965, Gates *et al.,* 2000, Brickner *et al.,* 2005**.

Treatment modalities to prevent noise induced hearing loss

Various preventive measures can be followed to eliminate the ill effects of noise exposure such as:

1. Introducing much improved automobile technology like masking of engine noise, sound proofing vehicles.
2. Wearing hearing protectors such as ear muffs and ear plugs.
3. Limiting periods of exposure to noise.
4. Looking for a noise rating while buying recreational equipment, children toys, household appliances and power tools.

Newer Treatment modalities :

Antioxidants that exhibit a protective effect against noise induced hearing loss were N-acetyl cystiene, acetyl –L- carnitine and vitamin-C. **Hamernik *et al*., 2008.**

Intracellular stress pathway inhibitors, Src-PTK inhibitors, are capable of preventing noise induced cell loss and hearing loss. **Harris KC, Hu B *et al.,* 2005.**

Glutaminergic neurotransmission blocker Riluzole, and Glutamate receptor antagonist caroverine have protective effect on hearing. **Wang J, Dib M *et al.,* 2002.**

Neurotropic factors, calcium channel blockers (T-type) have been found to be effective against noise induced hearing loss.

Recently the stem cells have been used in inner ear research in the hope that they will ultimately differentiate into hair cells and auditory neurons. **Parker MA, Corliss DA *et al.,* 2007.** These cells may be directed towards a hair cell or a neuronal phenotype to replace degenerated hair cells and neurons and finally restore inner ear function.

MATERIALS AND METHODS

A cross sectional descriptive study was designed with 50 subjects who are exposed to noise above the permissible limit and 50 controls who are exposed to noise within permissible limits. Both of these two groups selected were adult males and of the matching age group.

Study Group:

Noise exposed group comprised of Public transport drivers working at TamilNadu State Transport Corporation, Madurai. The selected Professional drivers were exposed to traffic noise due to their occupation as driving public transport bus in Madurai. They normally work for a period of 8-10 hours per day for 6 days in a week, with minimum 8 years of occupational noise exposure. During their working time they are exposed to the sound levels which are well above the WHO recommended noise exposure level.

Inclusion criteria:

Drivers of public transport, having minimum of 8 years of driving experience with minimum of 8 hours of driving per day. The age group was between 30-50 years.

Control group was selected from Office workers who work for about 8 hours per day, for 5-6 days in a week. Their noise exposure level was within permissible limit. The age group was between 30-50 years.

Exclusion criteria (for Both cases and controls):

Diabetes mellitus, Hypertension, H/o Ototoxic drugs, Middle ear disease like CSOM, Otosclerosis and Head injury.

Written informed consent was obtained from each subject after their requirements for participation in the study was explained. The study was preapproved by the ethical committee of our institution review board.

Methods:

A questionnaire was used to record the details of the subjects. Their age, education, years of driving, hours of driving, number of days of working per week were recorded.

The anthropometric measurements including height and weight were taken using inch tape and standard weighing machine.

Resting Blood pressure (B.P) was measured in all the subjects after they were seated for 10 minutes by using sphygmomanometer in right upper limb.

Clinical examination of the ear was done by an Otorhinolaryngologist which included examination for the presence of cerumen in the ear, structural assessment, mobility of the tympanic membrane, and abnormality of external auditory canal.

Rinne's test was done in all the subjects to compare air conduction with bone conduction of each ear separately. It is done by placing the base of the vibrating tuning fork over the mastoid process and the subject is asked to raise the hand. When he stops hearing the sound, then the vibrating tuning fork is transferred to the side of the head close to the meatus.

Weber's test was done in all the subjects to study the bone conduction. Vibrating tuning fork is placed on the middle of the forehead and the subject is asked to indicate in which ear the sound is heard better, or heard equally in both ears or in the centre of forehead. This is expressed as lateralization of sound to a particular ear or not.

After a detailed clinical history and examination of the ear, pure tone audiometry was performed for both Professional drivers and Office workers at the Institute of Physiology, Madurai Medical College, Madurai in a sound proof room after explaining about the test to the

subject. Since this is a subjective test it was started after getting the full cooperation of the subject.

Hearing examination included pure tone air and bone conduction audiometry. Audiometric testing was conducted according to the guidelines of the American Speech Language Hearing Association in sound treated room using EDA-3 N 3-MULTI Diagnostic Audiometer, calibrated according to ANSI (ANSI S3 1989, ISO 389 1991, IEC 645) and equipped with TDH-39 P ear phones.

Air conduction was tested with the better ear first with 1 kHz and then other frequencies were tested in the following order of 2, 4, 6, 8 kHz 500 and 250 Hz. First the subject is made to familiarize to the tone by introducing it at an arbitrarily presumed suprathreshold level. Then slowly the intensity of the tone is decreased until the subject no longer hears the sound. In this procedure the tones are lowered in 10 dB steps and increased in 5 dB steps.

The intensity at which the subject hears the tone for 50% of the time is marked as the **threshold of hearing** at that frequency. The second ear was tested in a similar manner and the hearing threshold was obtained for both ears by this conventional **Hughson-Westlak** technique and expressed in decibel hearing level units (dB HL).

Bone conduction was tested in the subject by placing the bone conduction vibrator with specification of Measurement of Bone vibrators (ANSI S3-13-1987) American National Standards Institute, 2002 over the mastoid in the same 5 up and 10 down method for all frequencies. In this study masking was not required.

The results obtained were graphically represented and the audiometric symbols presented in the Guidelines of **American Speech Language - Hearing Association, 1990** were used conventionally for plotting the pure tone audiogram.

Modality	**Right ear**	**Left ear**
Air conduction unmasked	○	X
Air conduction masked	△	□
Bone conduction unmasked	<	>
Bone conduction masked	[	]
No response		

The hearing threshold values obtained from the audiogram was interpreted by following WHO grades of hearing impairment.

Degree of hearing loss

Mild	26-40 dB
Moderate	41-55 dB
Moderately severe	56-70 dB
Severe	71-91 dB
Profound	>91 dB

The **pure tone averages** for the mid frequencies 500, 1000 and 2000 Hz and for the high frequencies 4, 6, and 8 kHz were calculated for both right and left ears.

The High frequency average is the criteria used to evaluate the function of hearing, since it is the frequency range known to be damaged maximally by excessive environmental noise and it is a region in which the effects of noise exposure first appear.

The most common configuration of Noise induced hearing loss is a bilateral, symmetrical sensorineural hearing loss with a notch at 4kHz in the audiogram **Alberti 1977.** A discrete loss of hearing for particular frequencies can result from damage to a part of the Organ of Corti e.g. exposure to intense sound, **Berne &Levy 6th edition.**

The reason for the occurrence of notch at 4 kHz:

1. Anatomical location of 4 kHz area of the basilar membrane corresponds to that area of basal turn of cochlea where it is firmly attached and therefore more prone for torsion and pressure changes in the perilymph.
2. Cochlea is more prone for vascular injuries in this area.
3. Reflex contraction of intratympanic muscles in response to loud sounds shift the sound towards higher frequencies.
4. Due to increased resonance of external auditory meatus, there is an increase in amplitude of sound waves in this frequency level.

Data Analysis:

The results were analysed to estimate the prevalence of noise induced hearing loss by observing the presence of the characteristic notch at 4 kHz. None of the subjects (cases & controls) in this study were having hearing threshold level above 70 dB. Hence hearing threshold levels upto 70 dB (0-25 dB, 25.1-40 dB, 40.1-55 dB and 55.1-70 dB) were included and analysed both in cases and controls.

RESULTS AND OBSERVATIONS

Mean (SD) age for the cases and controls were 39.38 ± 5.36 and 39.88 ± 4.92 years respectively. The mean (SD) height for the cases and controls were 163.08 ± 6.29 and 161.42 ± 7.24 cm respectively. The mean (SD) weight for the cases and controls were 65.88 ± 5.98 and 62.38 ± 8.46 respectively.

The mean (SD) systolic B.P for the cases and controls was 129.02 ± 8.45 and 130.12 ± 9.59 respectively. The mean (SD) diastolic B.P for the cases and controls was 81.08 ± 6.02 and 78.72 ± 5.65 with statistically significant value of < 0.05.

Rinne's test was positive in all the subjects (cases & controls) i.e. air conduction was better than bone conduction. Weber's test was centralized i.e. heard equally on both sides in all the subjects (cases & controls).

Results of the analysis are presented in the following table

Variables	Cases n=50 Mean (SD)	Controls n=50 Mean (SD)	P value
Age (years)	39.38 (5.36)	39.88 (4.92)	0.628
Height (cm)	163.08 (6.29)	161.42 (7.24)	0.224
Weight (kg)	65.88 (5.98)	62.38 (8.46)	0.019
Systolic B.P (mm Hg)	129.2 (8.45)	130.12 (9.59)	0.612
Diastolic B.P (mm Hg)	81.08 (6.02)	78.72 (5.65)	0.046
Years of exposure to noise	13.70 (4.92)	10.54 (2.95)	<0.001
Hours of exposure to noise/ day	8.92 (1.31)	8.0 (0.0)	< 0.001
Days of exposure to noise/week	5.60 (0.86)	5.68 (0.47)	0.564
Mid frequency hearing thresholds Right ear	23.91 (6.60)	18.09 (7.39)	< 0.001
Mid frequency hearing threshold Left ear	23.76 (6.86)	18.15 (6.12)	< 0.001
High frequency hearing threshold Right ear	29.24 (8.08)	19.62 (6.90)	< 0.001
High frequency hearing threshold Left ear	29.67 (10.29)	19.93 (6.23)	< 0.001

P value < 0.05 is significant.

Table-1

Prevalence of Noise induced hearing loss (4 kHz notch) among cases and controls

4kHz notch	Cases		Controls	
	No.of cases	Percentage	No.of cases	Percentage
Present	32	64	4	8
Absent	18	32	46	92

Chi square - 31.64

P value - < 0.001

Among the cases, out of 50 persons 32 persons (64%) are found to have audiometric evidence of notch at 4 kHz, a feature characteristic of noise induced hearing loss, when compared to the controls where only 4 persons (8%) have a notch at 4 kHz and this is statistically significant with $p < 0.001$ by chi square test.

Table-2

Comparison of Mid frequency hearing threshold of both ears between cases and controls

Hearing threshold (dB)	Right Ear		Left Ear	
	Case	Control	Case	Control
0 – 25	32	44	30	42
25.1 – 40	18	5	18	7
40.1 – 55	0	1	2	1
55.1 – 70	0	0	0	0
Mean	23.91	18.09	23.76	18.15
SD	6.60	7.39	6.86	6.12
P value	< 0.001 significant		< 0.001 significant	

The mean (SD) hearing threshold levels of cases and controls at audiometric test frequencies of 0.5, 1 and 2 kHz for the right ear are 23.91 $\pm$ 6.60 and 18.09 $\pm$ 7.39 respectively, and for the left ear are 23.76 $\pm$ 6.86 and 18.15$\pm$ 6.12 respectively which is statistically significant with $p < 0.001$. In both ears the mid frequency hearing threshold is higher in cases when compared to the controls.

Table-3

Comparison of High frequency hearing threshold of both ears between cases and controls

Hearing threshold (dB)	Right Ear		Left Ear	
	Case	Control	Case	Control
0 – 25	19	41	21	40
25.1 – 40	28	8	22	10
40.1 – 55	3	1	6	0
55.1 – 70	0	0	1	0
Mean	29.24	19.62	29.67	19.93
SD	8.08	6.90	10.29	6.23
P value	< 0.001 significant		< 0.001 significant	

The mean (SD) hearing threshold levels of cases and controls at audiometric high frequencies of 4, 6 and 8 kHz for the right ear are 29.24 $\pm$ 8.08 and 19.62 $\pm$ 6.90 respectively and for the left ear are 29.67 $\pm$ 10.29 and 19.93 $\pm$ 6.23 respectively which is statistically significant with $p < 0.001$. In both ears the high frequency hearing threshold is higher in cases when compared to controls.

The values indicate that hearing damage of drivers are expected to occur sooner at high frequencies than at mid frequencies.

Table-4

Comparison of High and Mid frequency hearing thresholds of Right ear among Cases

Hearing threshold (dB)	Right Ear	
	High Frequency	Mid Frequency
0 – 25	19	32
25.1 – 40	28	18
40.1 – 55	3	0
55.1 – 70	0	0
Mean	29.24	23.91
SD	8.08	6.60
P value	< 0.001 Significant	

The mean (SD) high frequency average of right ear among Cases is 29.24 $\pm$ 8.08 which is higher when compared to mid frequency average 23.91 $\pm$ 6.60 and is statistically significant with $p < 0.001$.

Table-5

Comparison of High and Mid frequency hearing thresholds of Left ear among Cases

Hearing Threshold (dB)	Left Ear	
	High Frequency	Mid Frequency
0 – 25	21	30
25.1 – 40	22	18
40.1 – 55	6	2
55.1 – 70	1	0
Mean	29.67	23.76
SD	10.29	6.86
P value	0.001 Significant	

The mean (SD) high frequency average of left ear among Cases is 29.67 $\pm$ 10.29 which is statistically higher when compared to mid frequency average 23.76 $\pm$ 6.86 with significant value of $p = 0.001$.

Table-6

Comparing the prevalence of High and Mid frequency hearing loss among cases with 4 kHz notch

Hearing threshold dB	High frequency		Mid Frequency	
	No. of cases	Percentage	No. of cases	Percentage
< 25	4	8	12	24
> 25	28	56	20	40

Among the total of 32 persons who are having 4 kHz notch, 28 of them show increased hearing threshold of >25 dB in high frequencies with the prevalence rate of 56%. For mid frequency sounds, 20 of them show increased hearing threshold of >25 dB with the prevalence rate of 40%.

This coincides with the fact that higher frequency sounds are affected more and first in excessive noise exposure.

Table-7

Comparison of High and Mid frequency hearing thresholds among Cases with 4 kHz notch (Right ear)

Hearing Threshold (dB)	(Right Ear)	
	High Frequency	Mid Frequency
0 – 25	6	17
25.1 – 40	23	15
40.1 – 55	3	0
55.1 – 70	0	0
Mean	32.36	25.44
SD	7.45	6.58
P value	< 0.001 Significant	

Statistically significant increase in the hearing threshold values of 32.36 $\pm$ 7.45 for high frequency sounds are present in the drivers having a notch at 4 kHz when compared with the mid frequency hearing threshold values 25.44 $\pm$ 6.58 in the right ear with $p < 0.001$.

Table-8

Comparison of High and Mid frequency hearing thresholds among Cases with 4 kHz notch (Left ear)

Hearing Threshold (dB)	(Left Ear)	
	High Frequency	Mid Frequency
0 – 25	8	14
25.1 – 40	17	16
40.1 – 55	6	2
55.1 – 70	1	0
Mean	33.42	25.48
SD	9.69	7.46
P value	< 0.001 Significant	

Statistically significant increase in the hearing threshold values of 33.42 $\pm$ 9.69 are present in the Cases having a notch at 4 kHz for high frequency sounds when compared to the mid frequency hearing threshold values 25.48 $\pm$ 7.46 in the left ear with $p < 0.001$.

Table-9

Relation between years of driving and hearing Loss

(4 kHz notch)

Years of Driving	No. of cases	Present	Absent
< 10	16	9 (56%)	7 (44%)
10 – 20	28	18 (64%)	10 (36%)
> 20	6	5 (83.3%)	1 (16.7%)

Significant increase in noise induced hearing loss is evident in drivers with increase in the duration of noise exposure.

The drivers with < 10 years of noise exposure have 56% prevalence of 4 kHz notch, and the drivers with 10-20 years of exposure have 64% prevalence of 4 kHz notch. The drivers with > 20 years of exposure have a high prevalence of about 83 %. The percentage prevalence of 4 kHz notch increases with increasing years of noise exposure.

The hearing loss positively correlates with duration of exposure to noise. As the years of exposure to noise increases, the hearing threshold also increases linearly both in mid frequency and high frequency range in both ears among the cases.

Statistical analysis:

The comparison between the cases and the controls were done by using one–way ANOVA test using SPSS (Statistical Package for Social Science) software, Sigma stat version 3.5. The significance was drawn at p value (probability) of < 0.05.

DISCUSSION

Of the several studies carried out, a study by **Leonog S T, Laortanakul P 2003** revealed that among the occupational population, the driver groups were found to have the highest risk of traffic noise induced hearing loss. It has also been found that there is a selective sensorineural hearing loss which affects the high frequency sounds first and is seen as a notch at 4 kHz in pure tone audiogram. Outcome of my study is also equally comparable to their study.

According to **Mc Bride DI, and S.Williams**, **2001 Prevalence** of noise induced hearing loss was identified by the presence of a notch in either ear. Their study also confirmed that with exposure to steady noise, the first well established clinical and valuable sign in confirming the diagnosis was the notch in the audiogram, maximal at 4 kHz. In the present study also the increased prevalence of hearing loss in noise exposed group was identified by the notch at 4 kHz and 64% of the drivers showed this valuable sign in their audiogram.

Present study coincides with the work done **by Imitraz Siddique, Riaz Siddique, 2008** which showed significant difference in prevalence of hearing loss between the noise exposed and non noise exposed group.

Studies by **Lt Col S Nair, RC Kashyap 2009, and Pepe PE, Jerger S et al.,** on prevalence of hearing loss in noise exposed group observed statistically significant increase in hearing loss with increase in duration of noise exposure. In the present study also, the prevalence rate of hearing loss is high when compared with the controls and it increases with the increase in the years of noise exposure.

Present study showed statistically significant difference ($p<0.001$) in hearing threshold levels of both ears at mid and high audiometric frequencies between drivers and office workers. This finding is consistent with conclusions of the investigators **Sanders and Mc cormick, 1992; Prince *et al*., 1997**, **Joshi SK, Devkota S *et al.*, 2003.**

Present study showed significantly increased overall hearing threshold levels of Professional drivers as compared to Office workers. This finding is similar to that of the previous study done on **"Excess risk estimates of hearing impairment of Indian professional drivers,"** by **J. Majumder, C.R. Mehta, and D. Sen 2009.**

The present study showed the mean hearing threshold level of high frequencies for left ear was higher as compared to right ear in

Professional drivers although there was no statistically significant difference between both ears. The similar trend was observed by **Kumar *et al*., Mohsen Janghorbani, Siamek Pourabdian, 2009.** This might be a manifestation of a **lateral difference in susceptibility to noise** damage and the left ear is more susceptible, which coincides with the study by **Chung DY,Willson GN, Gannon RP, 1983, Job et al., 1998.**

According to the study by **Pirila T, Jounio-Ervasti K *et al*., 1992** a significant average inferiority of the hearing in the left ear was found at high frequencies. In the present study also we had a comparable result which showed reduced hearing sensitivity in the left ear, seen as increased hearing thresholds at high frequencies especially 4 - 8 kHz. This asymmetry may be attributed to the **more pronounced efferent auditory system on the right side** which reduces the susceptibility of the right ear to cochlear insult **Nageris BI, Raveh E *et al.,* 2007.**

Glorig 1954, Karlovich *et al.,* J.W. Thelin, D.J. Joseph *et al*.,1983, Regina P, Edina MK Silva *et al.,* 2008, reported that noise exposed persons had significantly more hearing loss over the high frequency range than office workers in the same age categories. We

have also got similar results showing higher prevalence of high frequency hearing loss in Professional drivers when compared to Office workers.

CONCLUSION

Present study has confirmed the findings of others that noise exposed personnel are at risk of hearing loss. The hearing loss is correlated with duration of noise exposure and the frequency of the sound exposed. The values of average hearing thresholds indicated that hearing damage was expected to occur sooner at high frequencies than at mid and low frequencies.

It may be concluded that occupational hazards of professional driving significantly increased the hearing threshold levels of drivers as compared to office workers. Type of occupation seems to be an important factor in determining the auditory threshold profile of the individuals.

In the last few years, progress has been developed in various potential therapeutic approaches in addition to the preventive measures. It has been shown that hair cell loss mediated by noise may be prevented by antioxidants, inhibitors of intracellular stress pathways, neurotropic factors, neurotransmission blockers, and T-type calcium channel blockers. Recently the stem cells have been used in inner ear research in the hope that they will ultimately differentiate into hair cells and auditory neurons.

Public awareness of the hazardous effects of noise is low. To emphasise on this, the fourth Wednesday of April every year has been declared “International Noise Awareness Day” (**INAD**). As part of INAD a “Quiet day” is encouraged by observing 60 seconds of silence from 2.15 p.m to 2.16 p.m.

The reduction, if not stopping of everyday noises around us will improve our hearing and reduce the unwanted effects of noise.

BIBLIOGRAPHY

1. Atlas of Human Anatomy. Frank H.Netter. 5th edition, 2006, Saunder's Elsevier Publications.
2. Axelsson A, Borchgrevink H, Henderson D, Hellstrom P. Scientific basis of noise induced hearing loss. Newyork Thieme, 1996.
3. Axelsson A. Diagnosis and treatment of occupational noise induced hearing loss. Acta otoiaryngol suppl 1979; 360: 86-87.
4. Ballenger's Otolaryngology Head & Neck Surgery. James B. Snow JR, P. Ashley Wackym. 17th edition, 2009, People's Medical Publishing House.
5. Barrenas M.L, Bratthall A, Dahlgren J. The association between short stature and sensorineural hearing loss. Hearing Research 2005; 205: 123-130.
6. Berne & Levy physiology. Bruce H.Koeppen, Bruce A. Stanton. 6th edition, 2009, Mosby Elsevier publications.
7. Best and Taylor's physiological basis of Medical practice. John B.West. 12th edition, 1990, B.I.Waverly pvt Ltd.
8. Bogoch I.I, House R.A and Kudla I. Perceptions about hearing protection and noise induced hearing loss of attenders of rock concerts. Canadian journal of public health 2005; 96: 69-72.

9. Bohne B.A, Yohman L, Gruner MM. cochlear damage following interrupted exposure to high frequency noise. Hear Res 1987; 29: 251-264.
10. Borchgrevink H. Does health promotion work in relation to noise? Noise Health, 2003; 5: 25-30.
11. Bredberg G. Cellular pattern and nerve supply of the human organ of corti. Acta Otolaryngol suppl 1968; 236: 1-135.
12. Chakraborty D.C, Mukherjee A. Status of road traffic noise in Calcutta Metropolis, India. Journal of Acoustic Society 1997; 2: 943-949.
13. Chung D Y, Wilson G N, Gannon R P. Lateral difference in susceptibility to noise damage. Audiology 1983; 22(2): 199-205.
14. Clark W W, Bohne B A. Effect of periodic rest on hearing loss and cochlear damage following exposure to noise. J Acoust soc Am 1987; 82: 1253-1264.
15. Clinical Audio-Vestibulometry for Otologists and Neurologists. Anirban Biswas. 3rd edition, 2002, Bhalani Publishing House.
16. Diseases of Ear, nose & throat. PL Dhingra, Shruti Dhingra. 5th edition, 2010, Elsevier Publications.

17. Diseases of the ear. Harold Ludman and Tony Wright. 6th edition, 1998, Arnold Publishers.
18. Essentials of Audiology. Gelfand S.A. 3rd edition, 2009, Thiemes Medical Publications Newyork.
19. Frank's J.R, Stephenson M.R, Mercy C.J. Preventing occupational hearing loss –A practical guide (NIOSH) publication 1996.
20. Ganong's Review of Medical physiology. Kim E.Barett, Susan M.Barman, Scott Boitano, Heddwen L.Brooks. 23rd edition, 2010, McGraw- Hill companies.
21. Gates GA, Couropmitree NN, Myers RH. Genetic association in age related hearing thresholds. Arch Otolaryngol Head and Neck Surgery June 1999; 125(6): 654-659.
22. Glorig A, and Nixon J. Hearing loss as a function of age. Laryngoscope 1962; 72: 1596-1610.
23. Gray's Anatomy. Susan Standring. 40th edition, 2008, Churchill Livingstone Elsevier Ltd.
24. Hackney CM, Mahendrasingam S, Penn A. The concentration of calcium buffering proteins in mammalian cochlear hair cells. Journal of Neurosci 2005; 25: 7867-7875.

25. Haiyan shen, Baoping zhang. Prophylactic and therapeutic functions of T-type calcium blockers against noise induced hearing loss. Hear Res 2007; 226(1-2): 52-60.

26. Hamernik RP, Qiu W, Davis B. The effectiveness of N-acetyl-L-cysteine in the prevention of severe noise induced hearing loss. Hear Res 2008; 239(1-2): 99-106.

27. Hansen HR, Bok J, Devalah AK, Zha XM, Green SH. Calcium calmodulin dependent protein kinase II and IV both promote survival but differ in their effects on axon growth in spiral ganglion neurons. Journal of neurosci Res 2003; 72: 169-184.

28. Harris KC, Hu B, Henderson D. Prevention of noise induced hearing loss with Src-PTK inhibitors. Hear Res 2005; 208(1-2): 14-25.

29. Hawkins J.E. The role of vasoconstriction in noise induced hearing loss. Ann Otol Rhinol Laryngology 1971; 80: 903-913.

30. Henderson D, Bielefeld EC, Harris KC. The role of oxidative stress in noise induced hearing loss. Ear Hear 2006; 27(1): 1-19.

31. Henderson D, Subramaniam M, Boettcher FA. Individual susceptibility to noise induced hearing loss Ear and Hearing loss 1993; 14:152-168.

32. Henderson, Deepak, Richard and Hamernik Roger. Noise induced hearing loss Basic mechanism, prevention and control. London NRN Publications 2001.

33. Hirose K, Liberman MC. Lateral wall histopathology and endocochlear potential in the noise damaged mouse cochlea. J. Assoc Res Otolaryngol 2003; 4: 339-352.

34. Hodgetts W, Szarko R, Reiger E. Influence of background noise & exercise on the listening levels I Pod users. International journal of Audiology 2009; 48: 825-832.

35. Hood JD, Poole JP, Freedman L. The influence of eye colour upon temporary threshold shift. Audiology 1976; 15: 449-464.

36. Imtiaz Ather Siddiqui, Riaz Ahmed Siddique. The effect of excessive noise exposure on the hearing thresholds of aviation workers in Karachi. Pak journal of med sci 2008; 24(4): 525-530.

37. Janssen N.A, Van vtiet P, Aarts F, and Brunekreef B. Assessment of exposure to traffic related air pollution of children attending schools near motorways. Atmospheric Environment 2001; 35: 3875-3884.

38. Job A, Grateau P, Picard J. Intrinsic differences in hearing performances between ears revealed by the asymmetrical shooting posture in the army. Hearing Research 1998; 122 (1-2): 119-124.

39. Johansson P, Hansson AC *et al*., NF- kappa B mediated glucocorticoid response in the inner ear after acoustic trauma. Journal of neuroscience Research. 2006; 83: 1066-1076.

40. Johnsson L.G, Hawkins JE. Degeneration patterns in human ears exposed to noise. Ann Otol Rhinol Laryngol 1976; 85: 725-739.

41. Joseph Sataloff. Medical Audiology Progress Report. Arch Otolaryngol 1965; 81(1): 103-107.

42. Joshi Sk, Devkota S, Chamling S, Shrestha S. Environmental noise induced hearing loss in Nepal. Kathmandu University Medical Journal 2003; 1(3): 177-183.

43. Karlovich, R. S, Wiley, T. L and Jensen D.V. Hearing sensitivity in farmers. American Hearing Association Nov 1981. Los angles.

44. Kim J, Morest DK, Bohne BA. Degeneration of axons in the brainstem of the chinchilla after auditory overstimulation. Hear Res 1997; 103: 169-191.

45. Konings A, Van Laer L, Van Camp G. Genetic studies on noise induced hearing loss: A Review Ear and hearing 2009; 30(2): 151-159.

46. Konings A, Van Laer L, Wiktorek-Smagur A et al., Candidate gene association study for noise induced hearing loss in two independent noise exposed populations. Ann Hum Genet 2009; 73(2): 215-224.
47. Kryter K D. The effects of noise on man. Academic press, Newyork, 1970; 491-492.
48. Kumar K, Jain V.K. A study of noise in various modes of transport in Delhi. Applied Acoustics 1994; 43(1): 57-65.
49. Le Prell CG, Yamachita D, Minami SB, Miller JM. Mechanism of noise induced hearing loss indicates multiple methods of prevention. Hear Res 2006.
50. Lefebvre PP, Malgrange B, Lallemend F, Van De Water TR. Mechanisms of cell death in the injured auditory system. Otoprotective strategies Audiol Neurotol 2002; 7: 165-170.
51. Leong S T, Laortanakul P. Monitoring and assessment of daily exposure of roadside workers to traffic noise levels in an Asian city. Environ Monit Assess 2003; 85(1): 69-85.
52. Liberman MC, Dodds M.C. Acute ultrastructural changes in acoustic trauma. Hearing Res 1987; 26(1): 45-64.
53. Lim D J, Dunn D E. Anatomical correlates of noise induced hearing loss. Otolaryngol clinics North America 1979; 12: 493-513.

54. Lopez I, Ishiyama G, Acuna D, Baloh RW. Immunolocalization of voltage gated calcium channel alpha1 subunits in chinchilla cochlea. Cell Tissue Res 2003; 313:177-186.

55. Lt col S nair, Air vice Mshl R C Kashyap. Prevalence of noise induced hearing loss in Indian Air force personnel. MJAFI 2009; 65: 247-251.

56. Lynch ED, Kil J. Compounds for the prevention and treatment of noise induced hearing loss. Drug Discovery Today 2005; 10: 1291-1298.

57. Majumder J, C.R. Mehta, D. Sen. Excess risk estimates of hearing impairement of Indian professional drivers. International Journal of Industrial Ergonomics 2009; 39: 234-238

58. Mc Bride and S. Williams. Air blast circuit breaker noise and hearing loss: A multifactorial model for risk assessment. Occup med 2000; 50(3): 173-181.

59. Medical physiology. Walter f.Boron, Emile L.Boulpaep. 2nd edition, 2009, Saunders Elsevier Publications.

60. Melnick W. Human temporary threshold shift and damage risk. Journal of acoust soc Am 1991; 90: 140-154.

61. Minami SB, Yamashita D, Schachl J, Miller JM. Calcineurin activation contributes to noise induced hearing loss. Journal of Neurosci Res 2004; 74: 383-392.

62. Mohsen Janghorbani phD, Ali shelkhi, Siamak Pourabdian M D. The prevalence and correlates of hearing loss in drivers in Isfahan, Iran. Arch Iranian med 2009; 12(2): 128-134.

63. Morata T.C. Promoting hearing health and the combined role of noise induced hearing loss and Ototoxicity. Audiological medicine 2007; 5: 33-40.

64. Morest DK, Kim J, Potashner SJ, Bohne BA. Long term degeneration in the cochlear nerve and cochlear nucleus of the adult chinchilla following acoustic stimulation. Micro Res Tech 1998; 41: 205-216.

65. Nageris B I, Reveh E. Asymmetry in noise induced hearing loss: relevance of acoustic reflex and left or right handedness. Neurotol 2007; 28(4): 434-437.

66. Nordmann A S, Bohne B A, Harding G W. Histopathological differences between temporary and permanent threshold shift. Hear Res 2000; 139: 13-30.

67. Otolaryngology. The Ear. Paparella and Shumrick. 2nd edition, 1980, W. B. Saunders Company.

68. Parker MA, Corliss DA, Grey B. Neural stem cells injected into the sound damaged cochlea migrate throughout the cochlea and express markers of hair cells, supporting cells, and spiral ganglion cells. Hear Res 2007; 232(1-2): 29-43.

69. Parks TN. The AMPA receptors of auditory neurons. Hear Res 2000; 147: 77-91.

70. Pirila T, Jounio- Ervasti K. Left- right asymmetries in hearing threshold levels in three age groups of a random population. Audiology 1992; 31(3): 150-161.

71. Pope C.A and Dockery D.W. Health effects of fine particulate air pollution. Journal of the air & waste management Association 2006; 56: 709-742.

72. Prince MM, Stayner L.T, Smith R.J, Gilbert S.H. A reexamination of risk estimates from the NIOSH occupational noise and hearing survey. Journal of the Acoustical society of America 1997; 101: 950-963.

73. Pujol R, Puel JL. Excitotoxicity, Synaptic repair and functional recovery in the mammalian cochlea. A review of recent findings. Ann NY Acad Sci 1999; 884: 249-254.

74. Pujol R. Sensitive developmental period and acoustic trauma noise induced hearing loss. Mosby, 1992; 196-203.

75. Quirk WS, Avinash G, Nuttall AL. The influence of loud sound on Red blood cell velocity and blood vessel diameter in the cochlea. Hear Res 1992; 63: 102-107.

76. Rabbitt K. D, Bohne B.A. Holes in the reticular lamina after noise exposure. Hear Res 1983; 11: 41-53.

77. Rabinowitz PM, Galusha D, Slade M D, et al., Audiogram notches in noise exposed workers. Ear Hear 2006; 27: 742-750.

78. Rabinowitz, H.D. Peter M, Noise induced hearing loss. American academy of family physicians 2000.

79. Rejina P, Edina M K, Silva, et al., Prevalence of high frequency hearing loss consistent with noise exposure among people working with sound systems and general population in Brazil. A cross sectional study. BMC Public health 2008; 8: 151.

80. S. Williams, Mc Bride D I. Audiometric notch as a sign of noise induced hearing loss. Occup Environ med 2001; 58: 46-51.

81. Saunders JC, Cohen YE, Szymko YM. The structural and functional consequences of acoustic injury in the cochlea and peripheral auditory system. Journal of Acoustic Society Am 1991; 90: 136-146.

82. Scott-Brown's Otolaryngology, Head and Neck Surgery Vol-3. Michael Gleeson. 7th edition, 2008, Edward Arnold Publications.

83. Slepecky N. Overview of mechanical damage to the inner ear noise as a tool to probe cochlear function. Hear Res 1986; 22: 307-321.

84. Taylor W, Pearson J, Mair A. Study of noise and hearing in jute weaving. Journal of Acoustic Society Ame 1965; 38: 113-120.

85. Textbook of Ear, Nose, Throat and Head & Neck surgery clinical & practical. P. Hazarika, D. P. Nayak, R. Balakrishnan. 2nd edition, 2009, CBS Publishers & Distributors.

86. Textbook of Medical physiology. Arthur C. Guyton, John E.Hall. 12th edition, 2006, Elsevier publications.

87. Vander's Human physiology. Iric P.Widmaier, Hersnel Raff, Kevin T.Strang. 11th edition, 2008, McGraw-Hill companies.

88. Wang J, Dib M, Lenoir M, Vago P. Riluzole rescues cochlear sensory cells from acoustic trauma in the guinea pig. Neuroscience 2002; 111(3): 635-648.

89. Wayne S. Quirk. Sarthran preserves cochlear microcirculation and reduces temporary threshold shift. Otolaryngol Head Neck Surg 1998; 118: 576-583.

90. Yamane H, Nakai Y, Jakayama M, et al., The emergence of free radicals after acoustic trauma and strial blood flow. Acta otolaryngology suppl 1995; 519: 87-92.

91. Yang M, Tan H, Yang Q. Association of hsp 70 polymorphisms with risk of noise induced hearing loss in Chinese automobile workers. Cell stress chaperones 2006; 11: 233-239.

92. Zannin P.H.T. Occupational noise in urban buses. International journal of Industrial Ergonomics 2008; 38: 232-237.

PROFORMA

Serial No. **Date :**

Name: **Age:** **Sex:**

Address:

Occupation : **Height :** **cm** **weight :** **kg**

Medical H/o: Diabetes/HT/Prolonged drug intake/hearing difficulty/ ear discharge

Personal H/o: Smoking Yes/No No. of years of smoking:

Alcoholic Yes/No No. of years of consumption:

No. of years of driving experience : No. of years of exposure to noise:

Type of Driving Vehicle:

No. of hours of exposure to noise per day:

No. of days of exposure to noise per week:

On Examination :

Pulse rate: **BP:**

Examination of ear : **Right** **Left**

External ear :

Tympanic membrane :

Rinne's Test :

Weber's Test:

Pure tone Audiometry

மருத்துவப் பரிசோதனை முறைகளைப் பற்றி மருத்துவரிடம் தெரிந்து கொண்டேன். இதனை மேற்கொள்ள நான்முழு மனதுடன் சம்மதிக்கிறேன்.

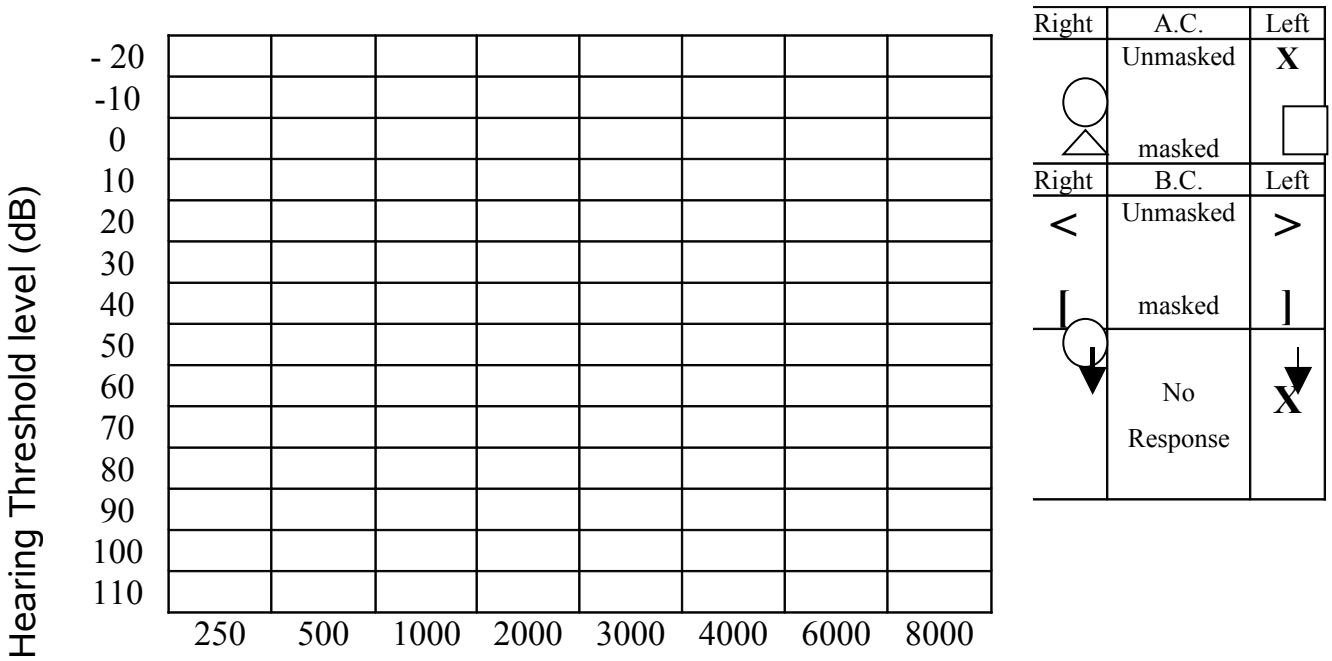
AUDIOLOGY AND SPEECH THERAPY SECTION

Hospital No. MRD No. Audio No. Date :

Name : Age : Sex :

Address :

Frequencies in Hz



Physiologist's Opinion :

Other Investigations :

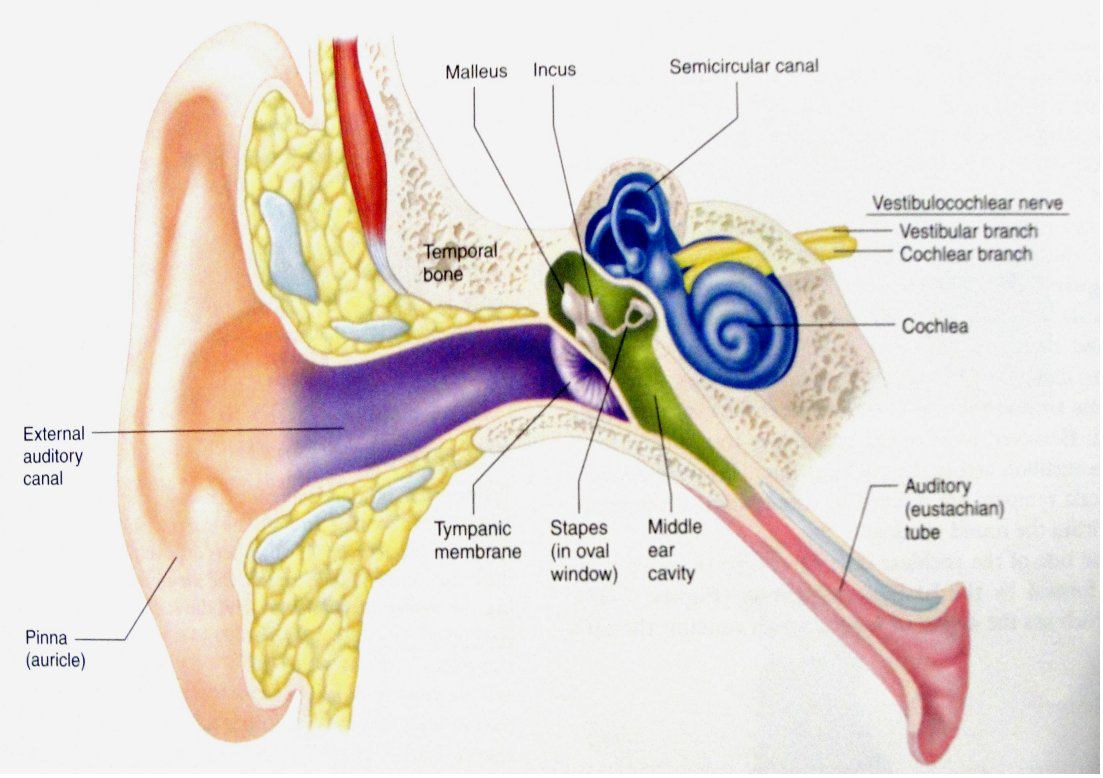
Provisional Diagnosis :

Advice :

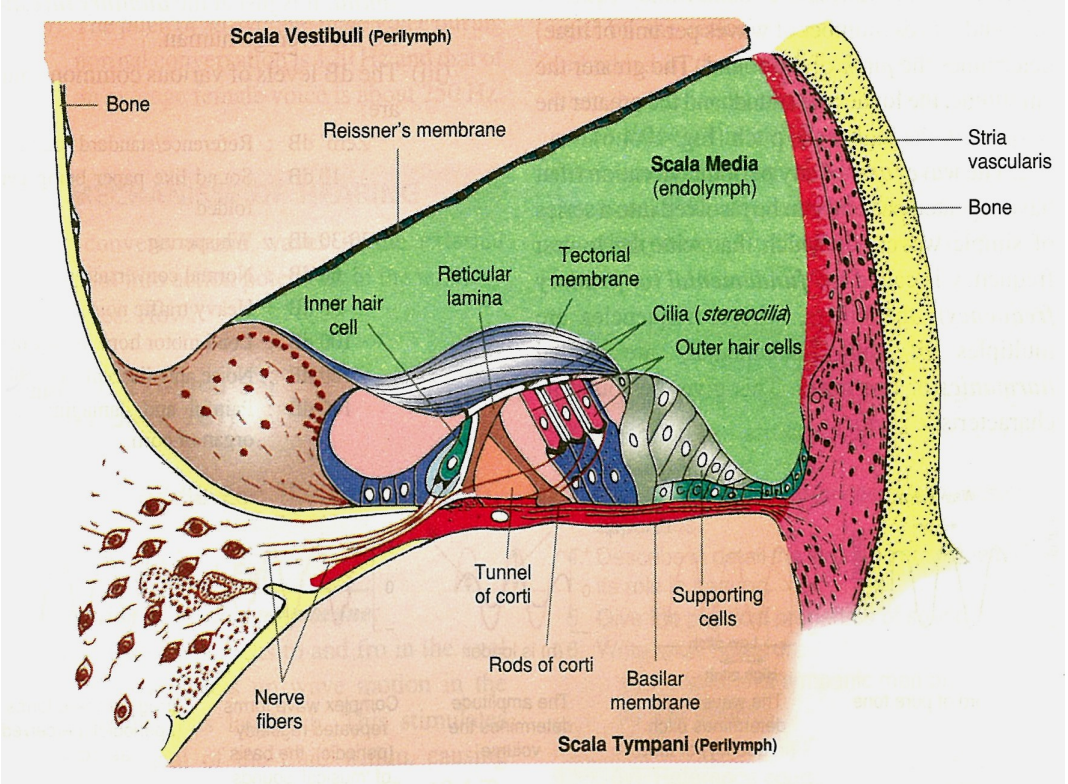
Follow up :

SPEECH THERAPIST SIGNATURE

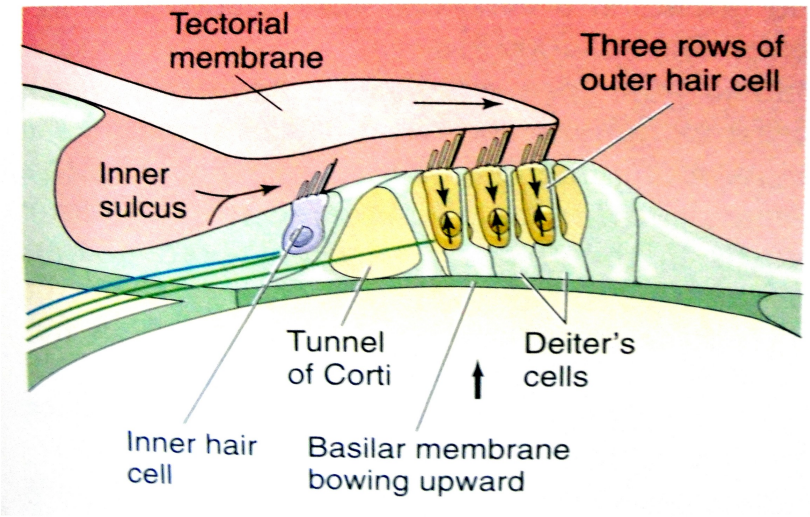
STRUCTURE OF HUMAN EAR



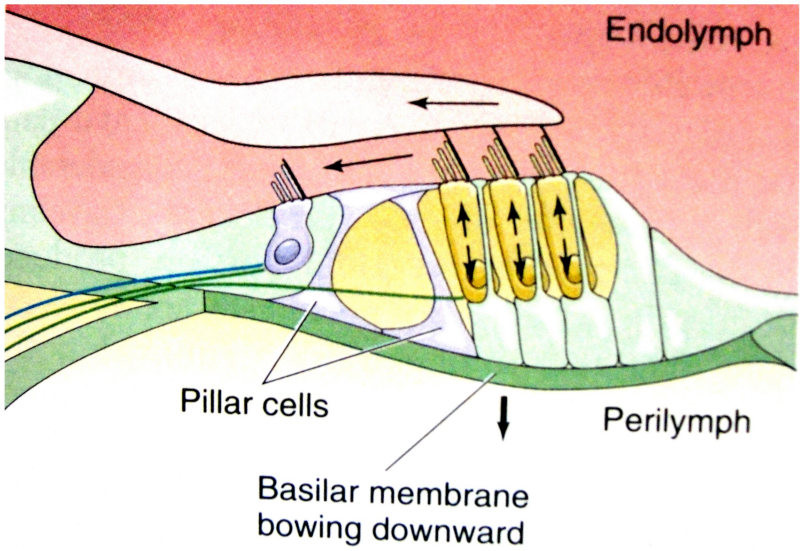
ORGAN OF CORTI



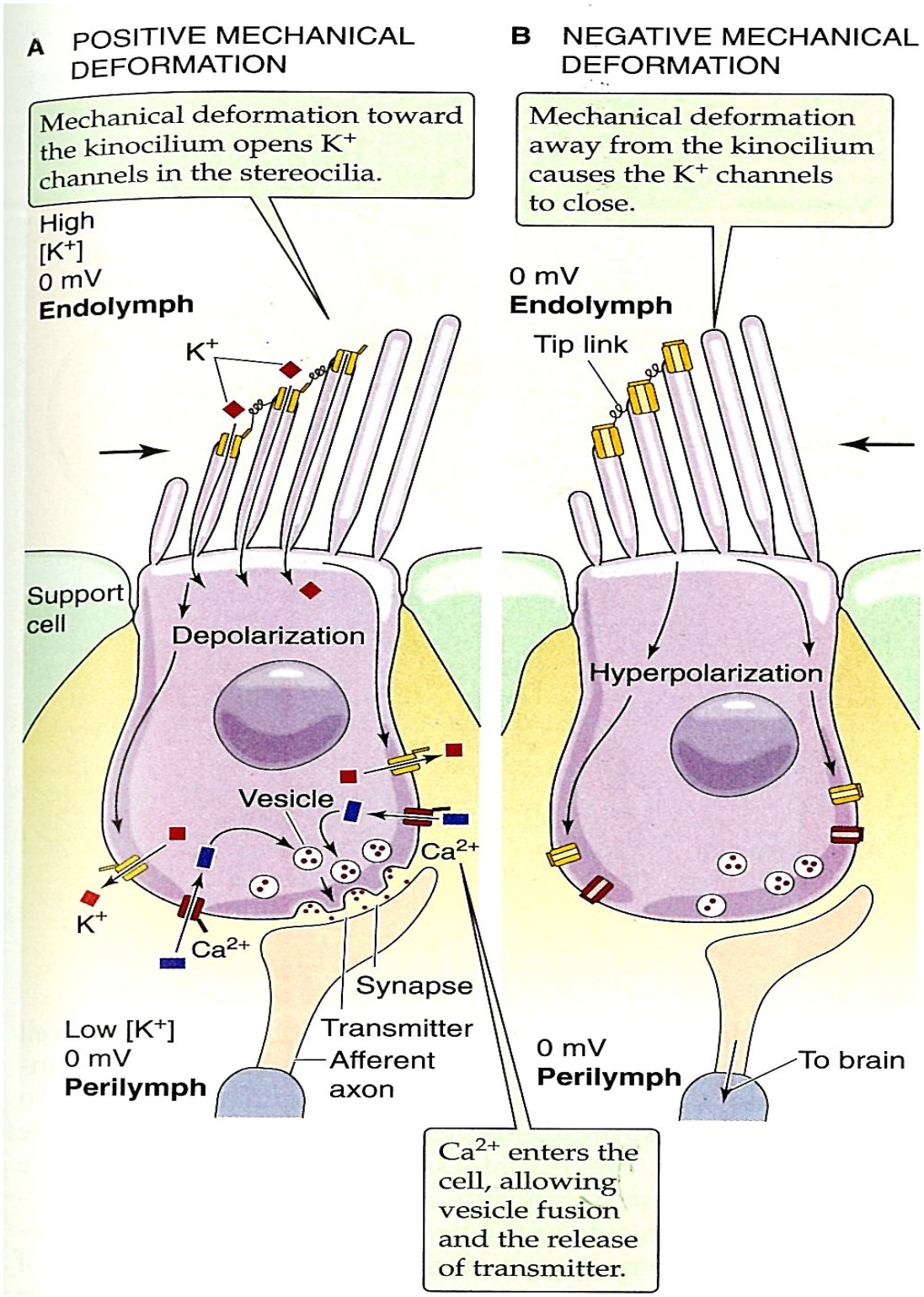
UPWARD MOVEMENT OF BASILAR MEMBRANE OPENING TRANSDUCTION CHANNELS



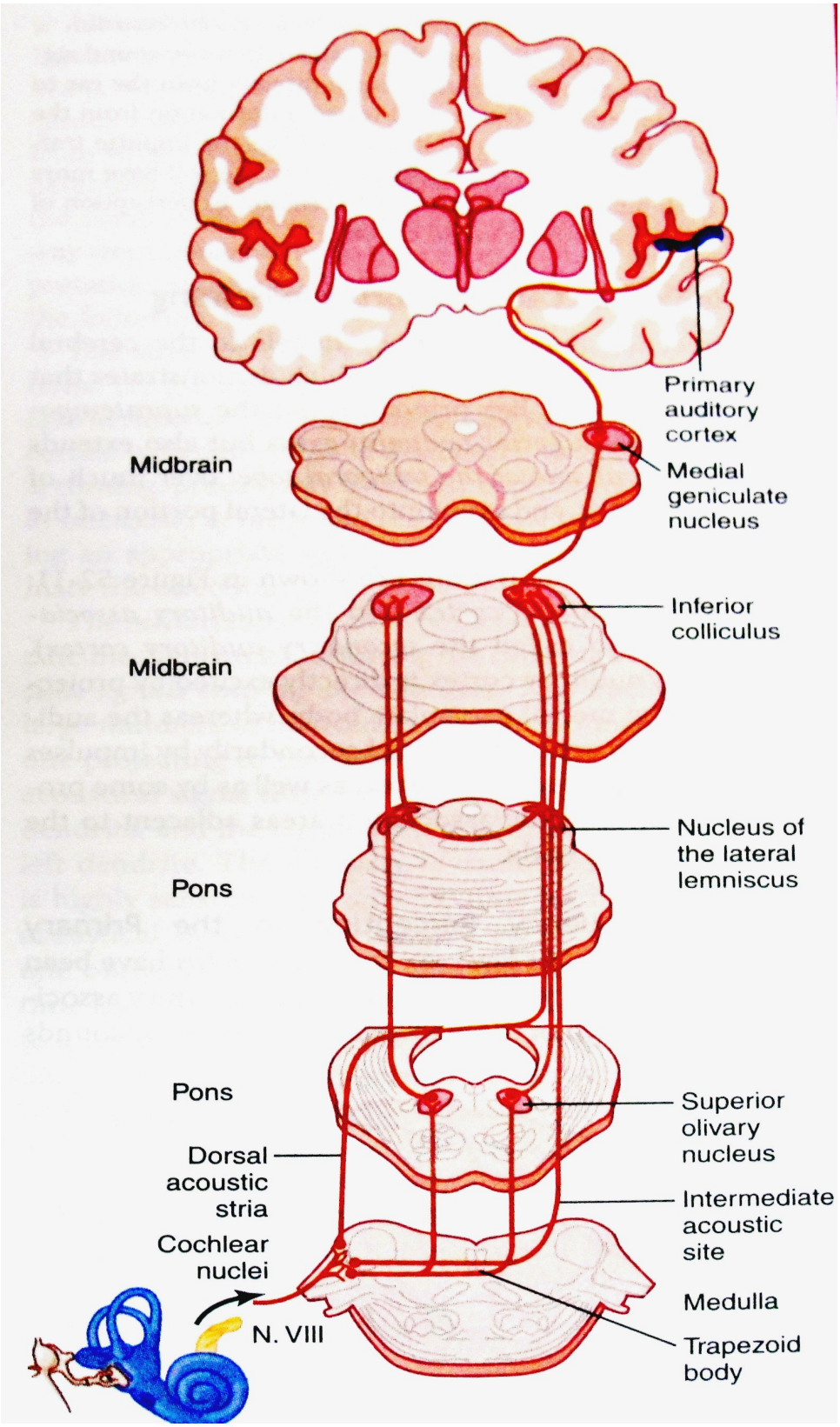
DOWNWARD MOVEMENT OF BASILAR MEMBRANE CLOSING TRANSDUCTION CHANNELS



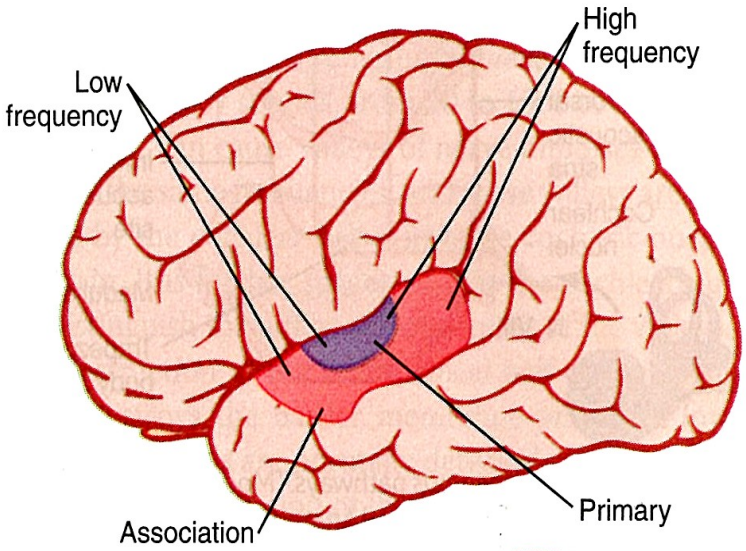
MECHANO TRANSDUCTION IN THE HAIR CELL



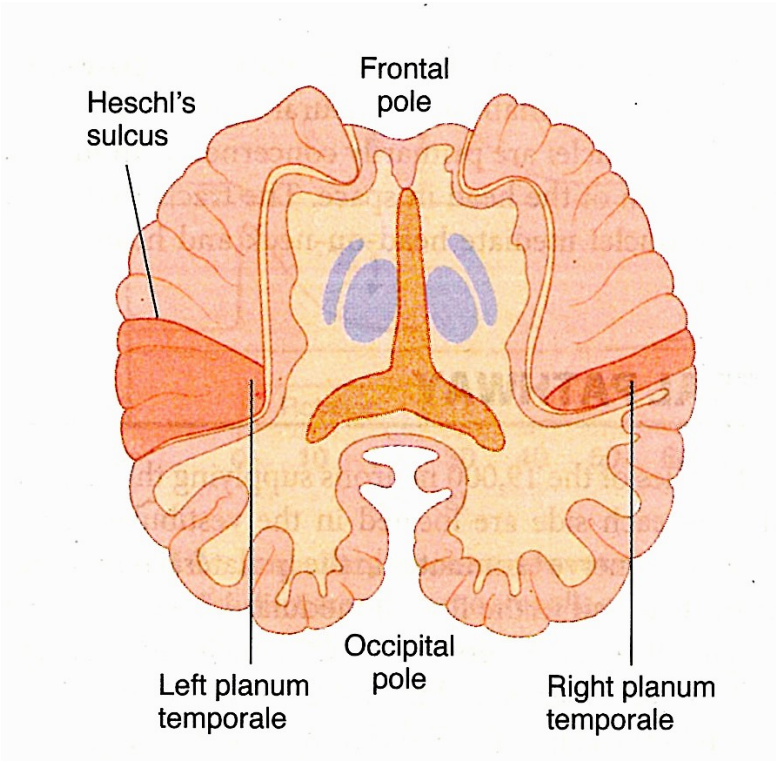
AUDITORY PATHWAY



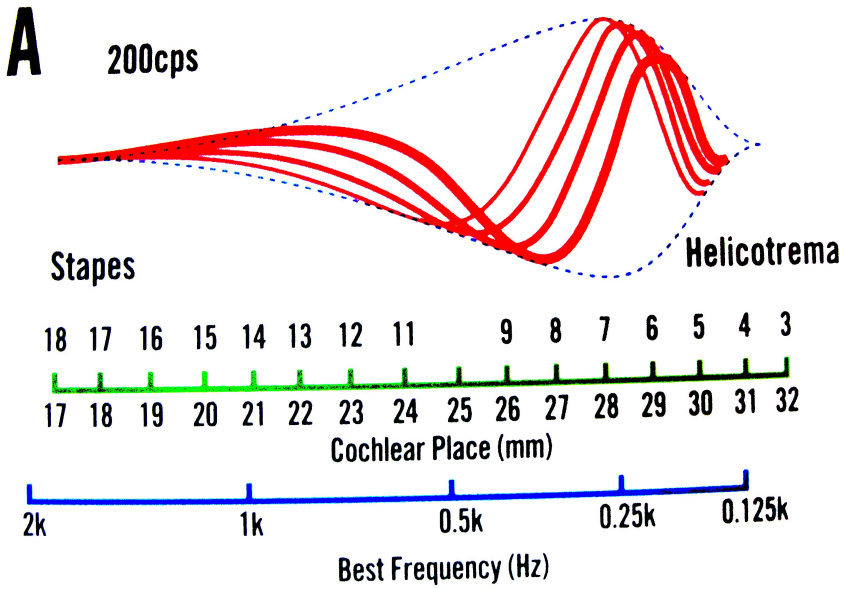
AUDITORY CORTEX

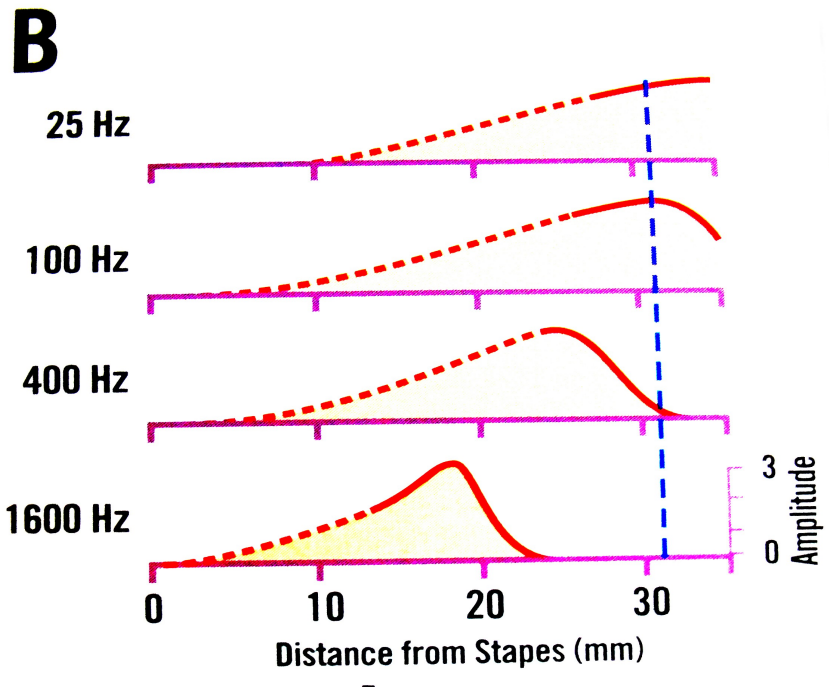


LANGUAGE RELATED AUDITORY PROCESSING AREA (PLANUM TEMPORALE)

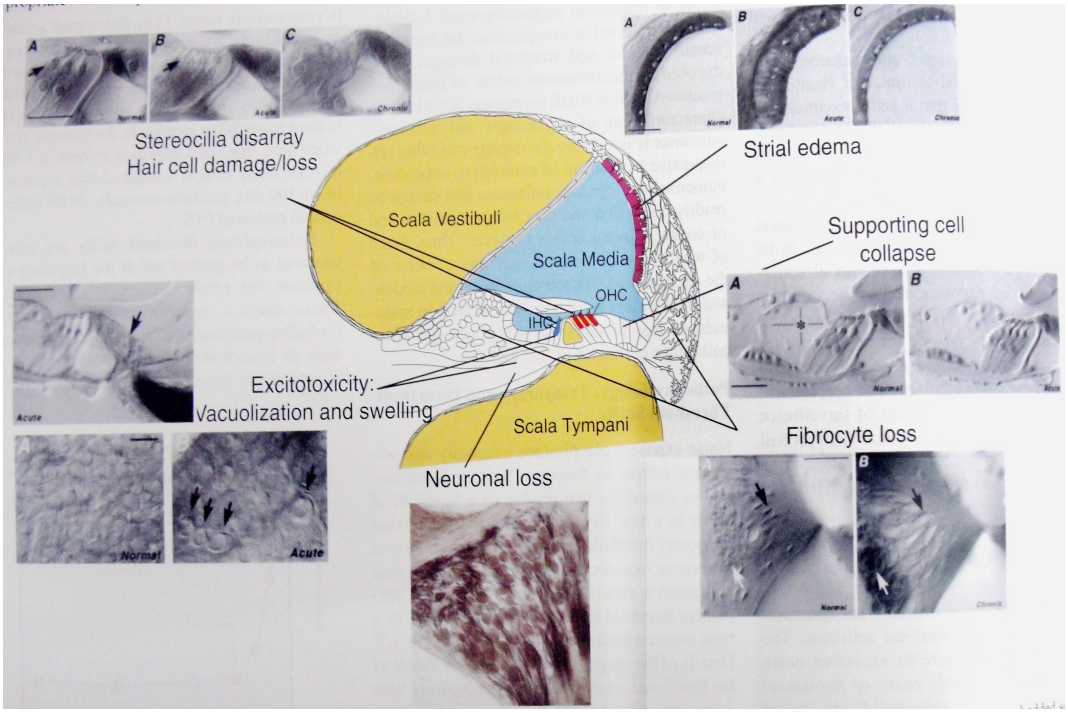


TRAVELLING WAVES FOR DIFFERENT FREQUENCIES OF SOUND

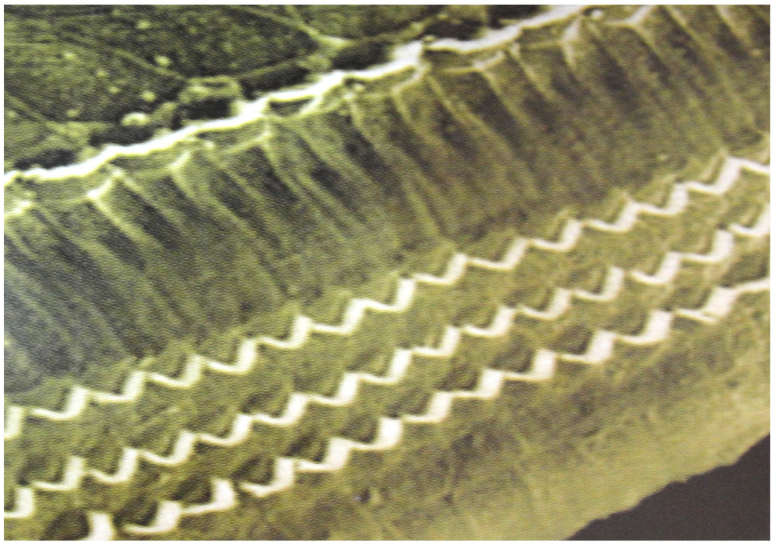




HISTOPATHOLOGICAL CHANGES IN ORGAN OF CORTI IN NOISE INDUCED HEARING LOSS



NORMAL HAIR CELLS OF THE COCHLEA



DAMAGED HAIR CELLS

RECORDING OF AUDIOGRAM

PURE TONE AUDIOMETER

4kHZ NOTCH IN AUDIOGRAM

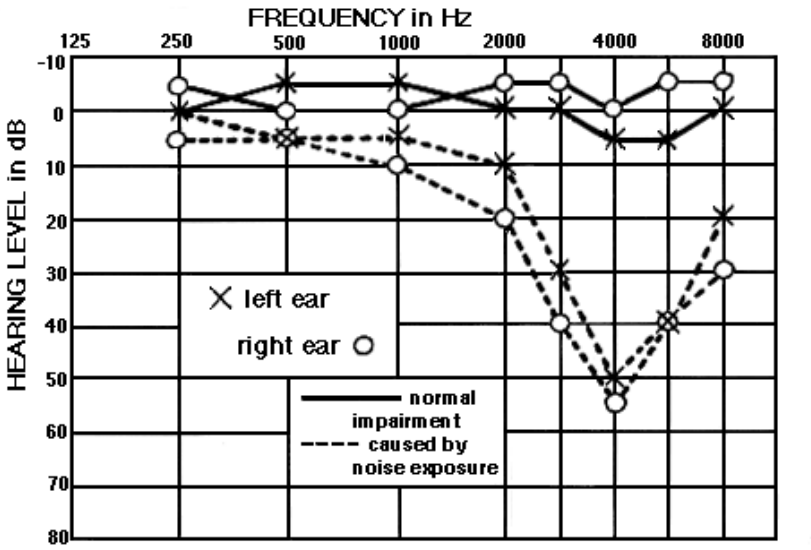


FIGURE - 1

Prevalence of noise induced hearing loss (4kHz Notch) among cases and controls

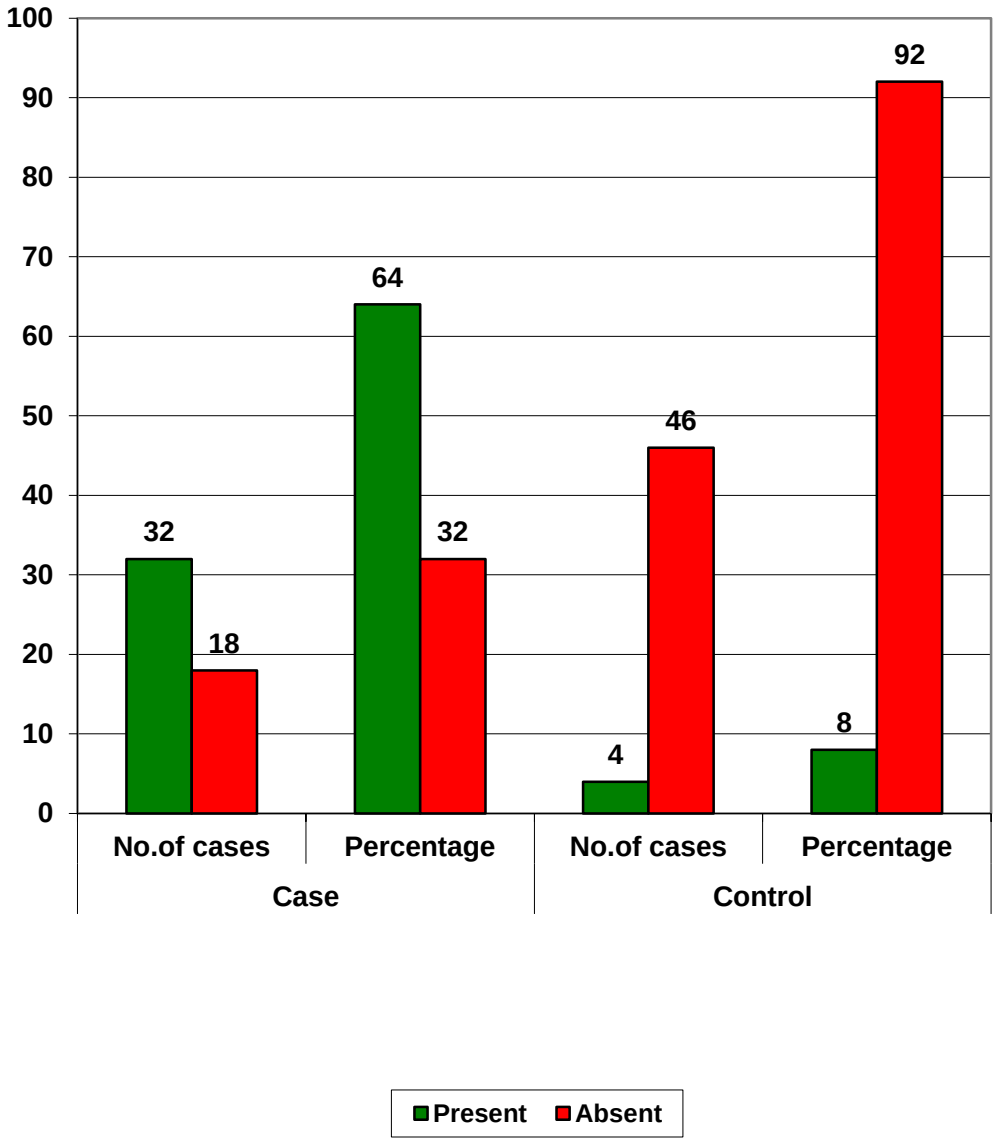


FIGURE - 2

Comparison of Mid frequency hearing threshold of both ears between cases and controls

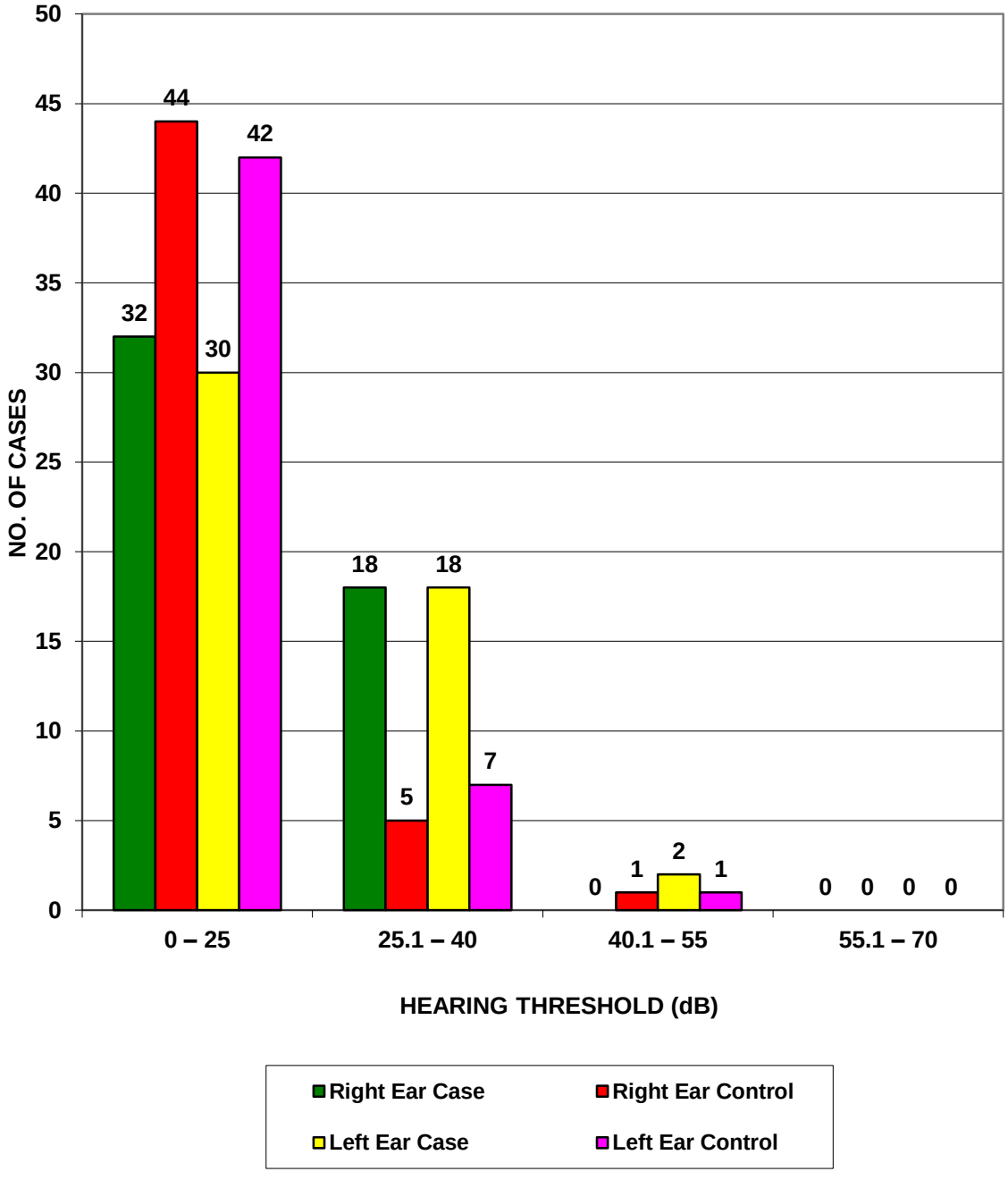


FIGURE - 3

Comparison of High frequency hearing threshold of both ears between cases and controls

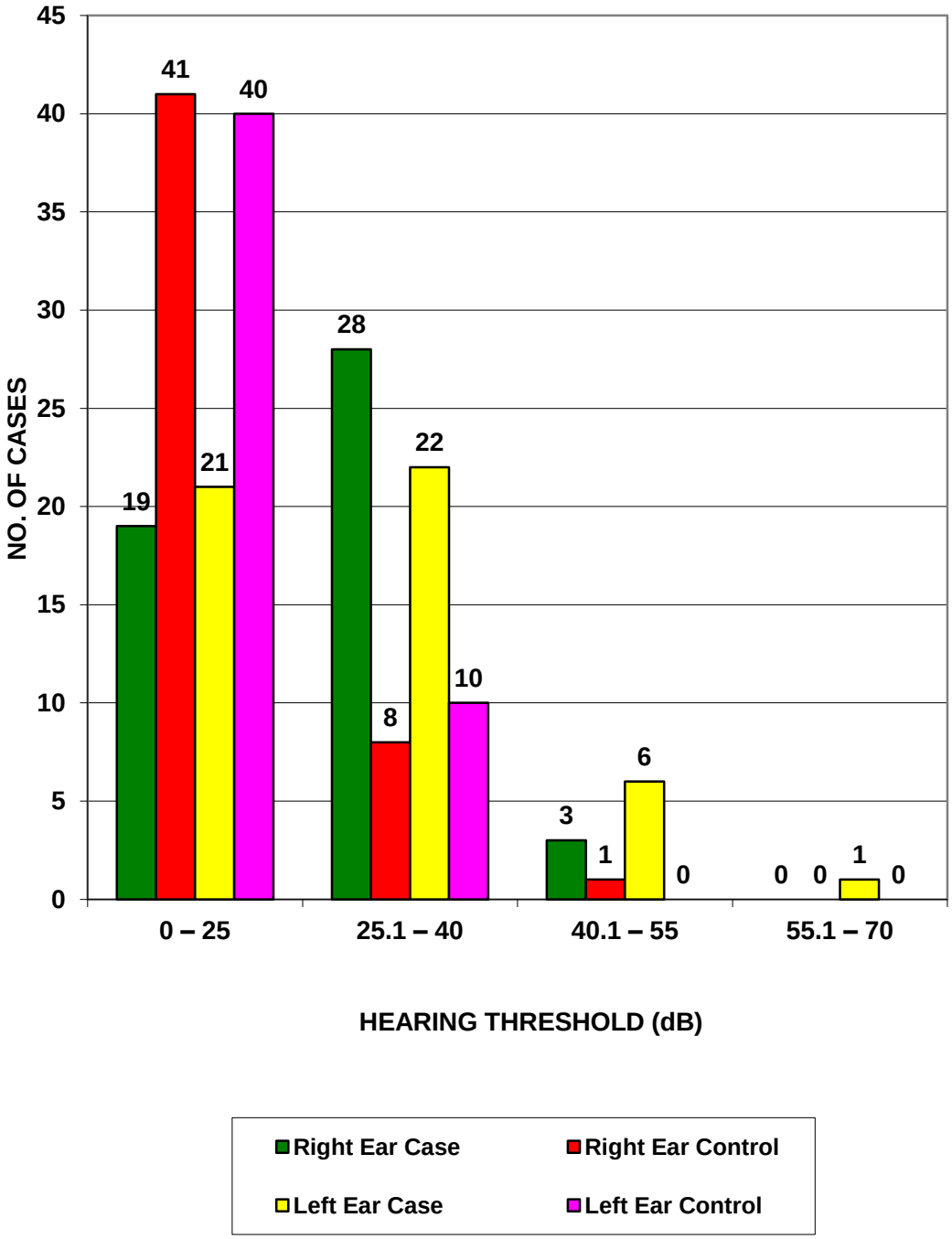


FIGURE - 4

Comparison of High and Mid frequency hearing thresholds of Right ear among Cases

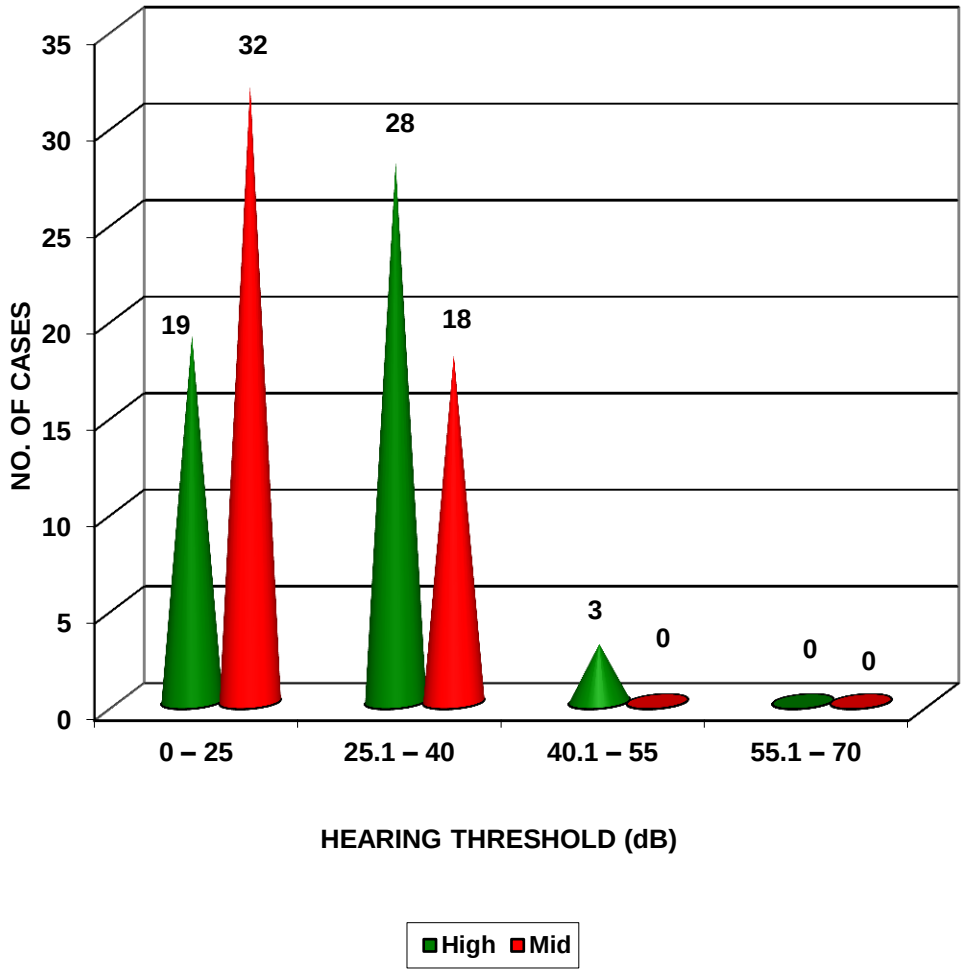


FIGURE - 5

Comparison of High and Mid frequency hearing thresholds of Left ear among Cases

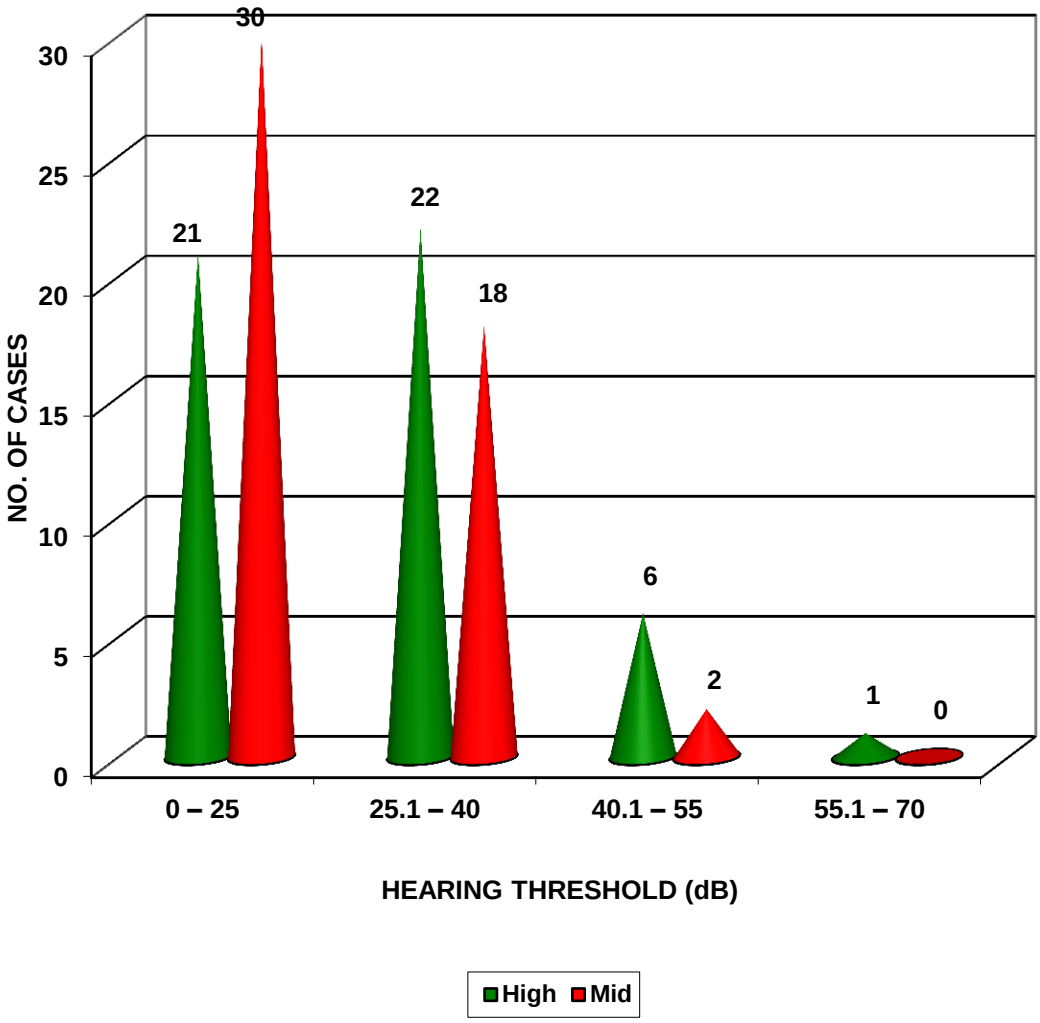


FIGURE - 6

Comparing the prevalence of High and Mid frequency hearing loss among Cases with 4 kHz notch

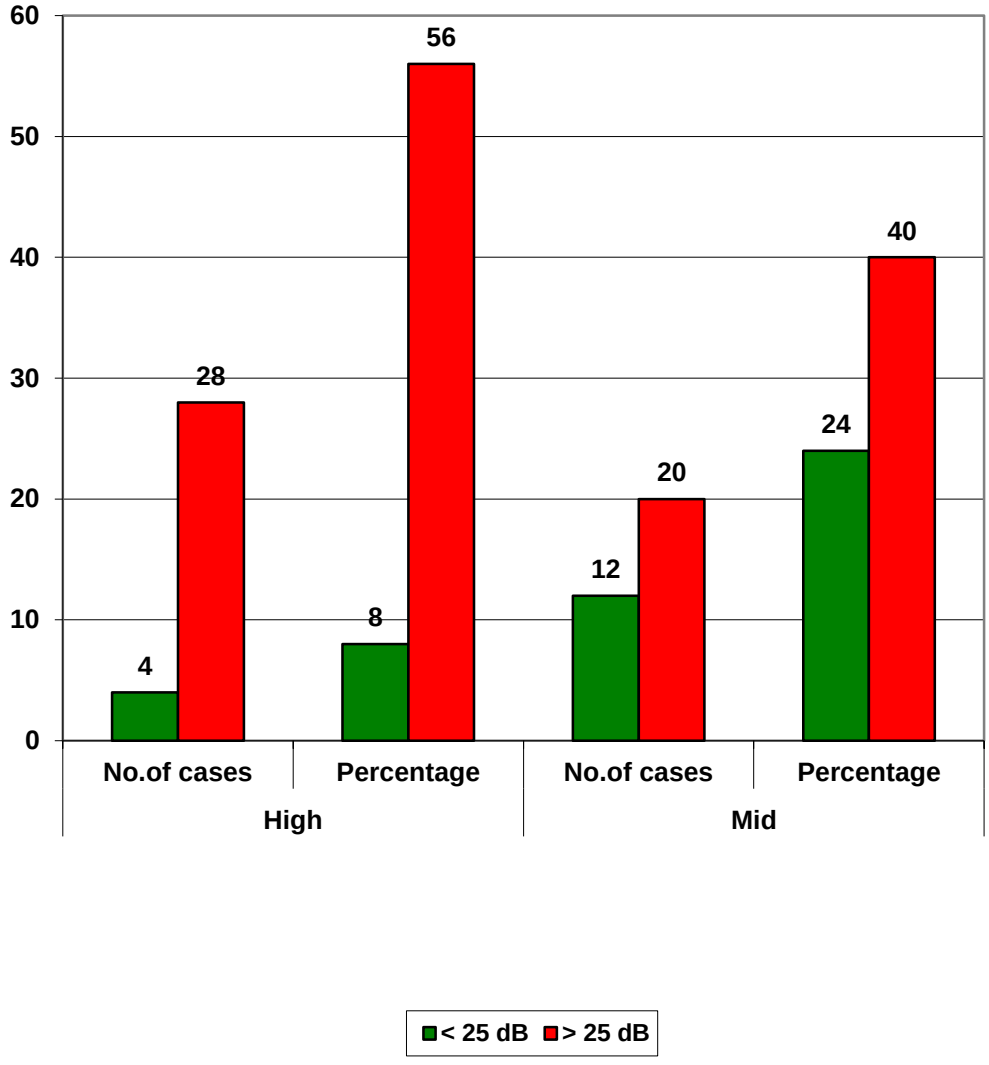


FIGURE - 7

Comparison of High and Mid frequency hearing thresholds among cases with 4 kHz notch (Right ear)

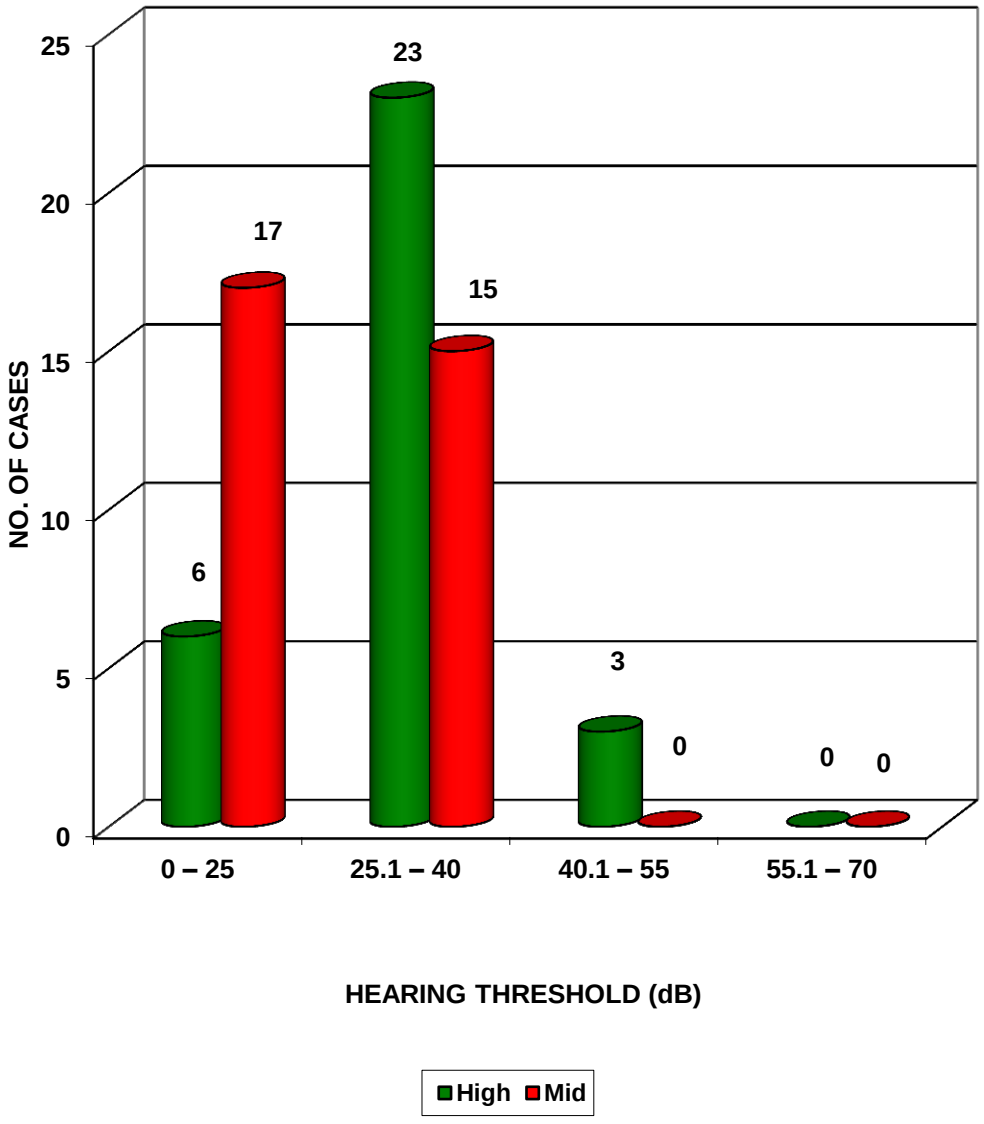


FIGURE - 8

Comparison of High and Mid frequency hearing thresholds among cases with 4kHz notch (Left ear)

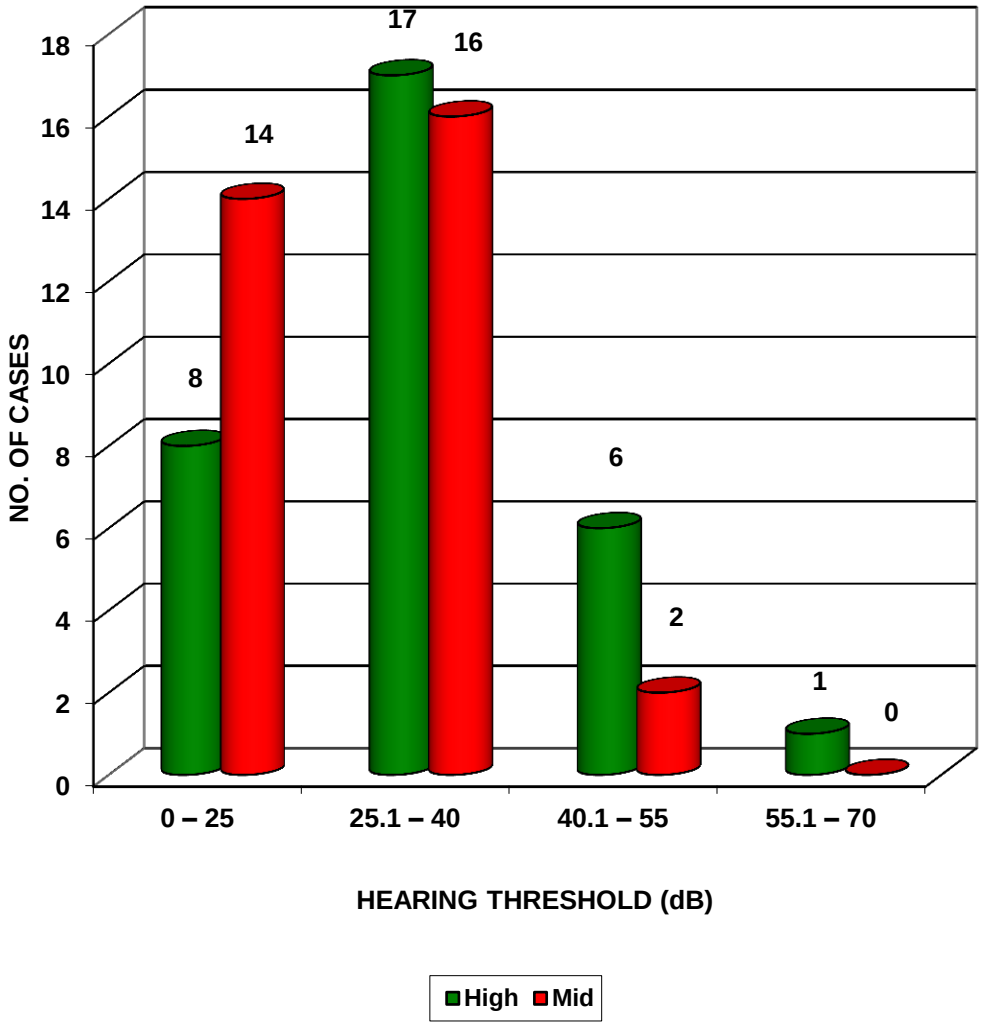


FIGURE - 9

Relation between years of driving and hearing loss (4 kHz notch)

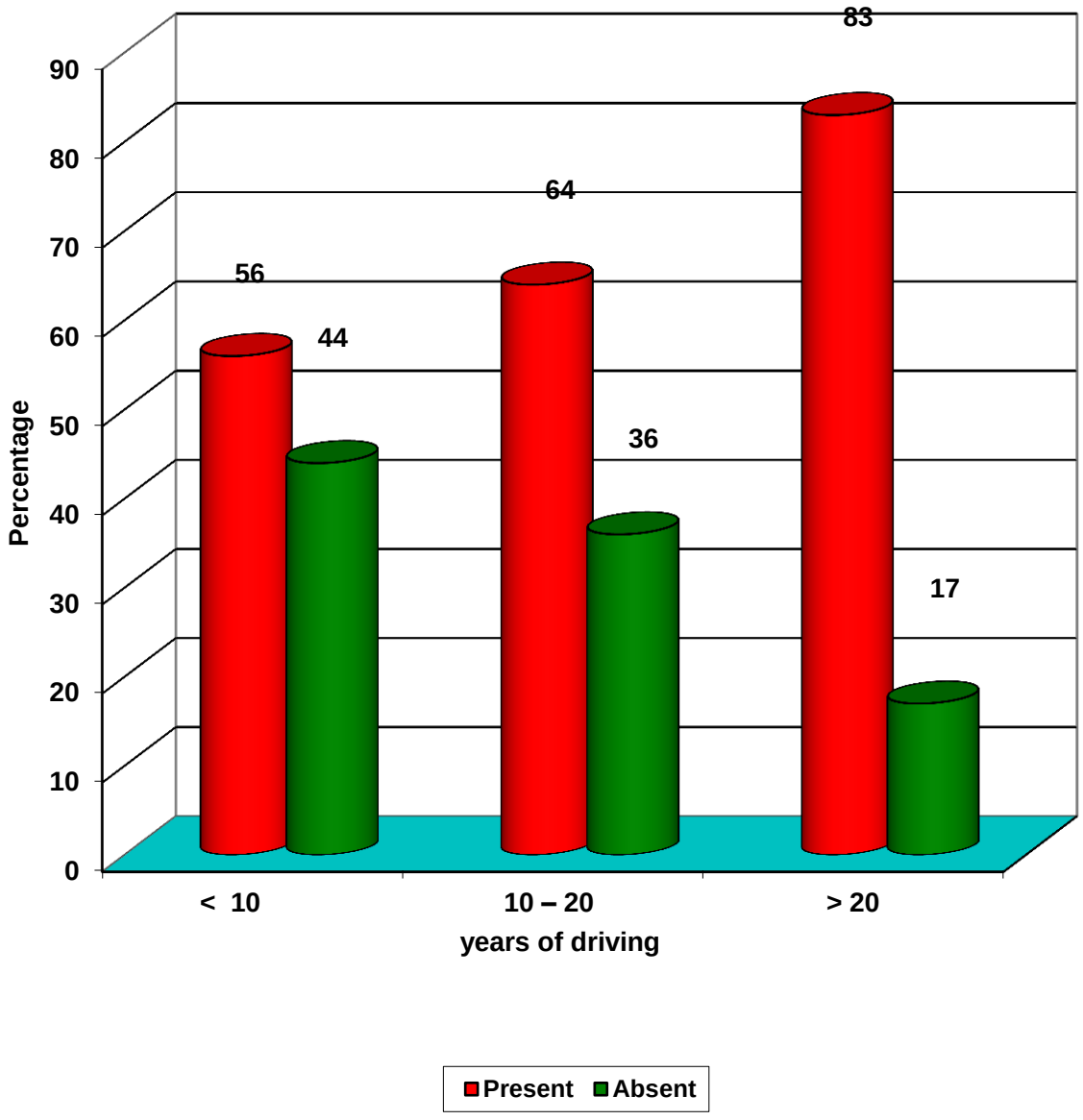


FIGURE - 10

Relation between years of exposure and hearing threshold for both frequencies in Right ear among cases

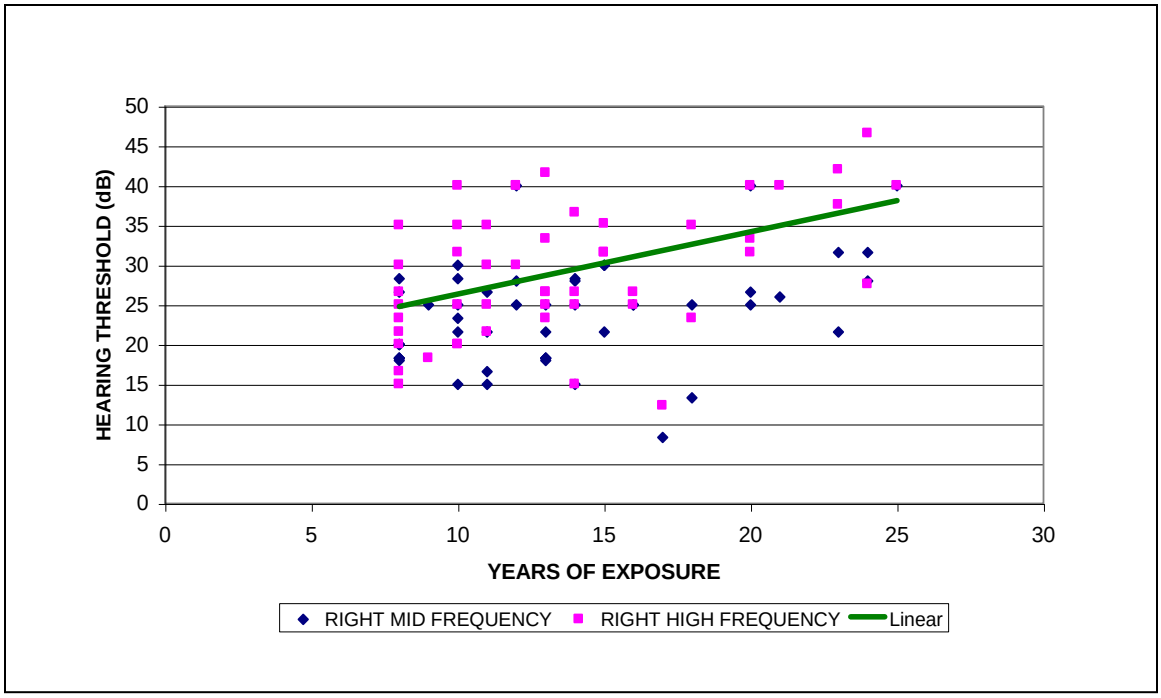
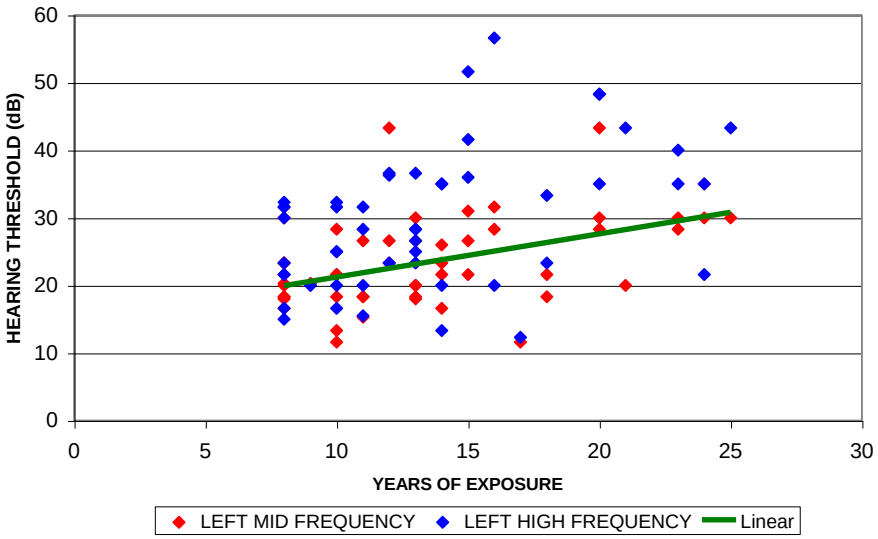


FIGURE - 11

Relation between years of exposure and hearing threshold for both frequencies in left ear among cases



MASTER CHART - CASES

S.No	Name	Age	Sex	Height (cm)	Weight (Kg)	Smoking	Alcoholic	Systolic BP	Diastolic BP	Type of vehicle	No. of years of driving	No.of years of exposure to noise	Hours of exposure to noise/ day	No. of days of exposure/ week	Decibel exposed	Rinnes test Right	Rinnes test Left	Weber's Test (Centralised)	PTA-4KHz Notch	4kHz Notch Right ear	4kHz Notch Left ear	Hearing threshold (Mid frequencies) Right ear (.5,1,2 kHZ Avg)	Hearing threshold (Mid frequencies) Left ear (.5,1,2 kHZ Avg)	Hearing threshold (High frequencies) Right ear (4,6,8 kHZ Avg)	Hearing threshold (High frequencies) Left ear(4,6,8 kHZ Avg)
1	Krishnan	49	M	164	62	No	No	130	70	B	18	18	8	6	Above 85	P	P	⟷	Absent	Absent	Absent	13.3	18.3	23.3	23.3
2	Sundaram	50	M	154	60	No	No	110	70	B	17	17	10	6	Above 85	P	P	⟷	Absent	Absent	Absent	8.3	11.6	13.3	11.6
3	Ramar	44	M	157	58	No	No	130	80	B	18	18	8	6	Above 85	P	P	⟷	Absent	Absent	Absent	25	21.6	35	33.3
4	Ayyanar	50	M	159	58	No	No	130	80	B	24	24	13	6	Above 85	P	P	⟷	Present	Present	Present	28.3	35	46.6	35
5	Murugan	40	M	163	65	No	No	120	70	B	21	21	9	6	Above 85	P	P	⟷	Present	Present	Present	26.6	20	40	43.3
6	Eswaran	35	M	157	63	No	No	110	70	B	13	13	8	6	Above 85	P	P	⟷	Present	Present	Present	18.3	26.6	33.3	26.6
7	Kathiresan	39	M	164	66	No	No	120	80	B	15	15	10	6	Above 85	P	P	⟷	Present	Present	Absent	30	31.6	31.6	41.6
8	Ravichandran	41	M	172	71	No	No	136	86	B	10	10	12	3	Above 85	P	P	⟷	Absent	Absent	Absent	23.3	21.6	20	16.6
9	Velusamy	39	M	158	62	No	No	140	80	B	8	8	8	6	Above 85	P	P	⟷	Present	Present	Absent	20	16.6	26.6	15
10	Karuppiah	38	M	158	64	No	No	130	70	B	8	8	8	4	Above 85	P	P	⟷	Present	Absent	Present	26.6	30	35	31.6
11	Senthilkumar	34	M	170	70	No	No	130	80	B	10	10	8	6	Above 85	P	P	⟷	Present	Present	Present	15	13.3	20	20
12	Santhanavel	39	M	155	60	No	No	120	70	B	13	13	8	6	Above 85	P	P	⟷	Present	Present	Present	18.3	18.3	26.6	25
13	Maharaja	33	M	172	68	No	No	130	70	B	13	13	10	6	Above 85	P	P	⟷	Present	Present	Present	21.6	20	41.6	28.3
14	Chellappan	37	M	157	60	No	No	134	84	B	8	8	9	6	Above 85	P	P	⟷	Absent	Absent	Absent	20	18.3	16.6	21.6
15	Joseph	44	M	160	72	No	No	140	86	B	14	14	9	6	Above 85	P	P	⟷	Present	Present	Present	15	16.6	26.6	35
16	Sangilimurugan	33	M	173	68	Yes	No	130	70	B	16	16	9	6	Above 85	P	P	⟷	Absent	Absent	Absent	25	28.3	25	20
17	Arumugam	50	M	164	65	Yes	No	140	90	B	24	24	8	6	Above 85	P	P	⟷	Absent	Absent	Absent	31.6	30	26.6	21.6
18	Veeraalagarsamy	37	M	165	78	No	No	130	80	B	8	8	10	6	Above 85	P	P	⟷	Absent	Absent	Absent	18.3	20	25	21.6
19	Damodarakannan	33	M	160	64	No	No	120	70	B	13	13	8	6	Above 85	P	P	⟷	Absent	Absent	Absent	18.3	20	23.3	23.3
20	Velmurugan	33	M	158	52	No	No	120	70	B	8	8	9	6	Above 85	P	P	⟷	Present	Absent	Present	20	23.3	15	23.3
21	Sethuraman	32	M	160	63	No	No	120	70	B	9	9	8	6	Above 85	P	P	⟷	Absent	Absent	Absent	25	23.3	18.3	20
22	Ochadevan	35	M	162	64	No	No	130	80	B	8	8	8	6	Above 85	P	P	⟷	Absent	Absent	Absent	18.3	18.3	20	16.6

23	Kuppapuli	38	M	166	66	No	No	136	84	B	13	13	8	6	Above 85	P	P	⟷	Present	Present	Absent	25	30	25	28.3
24	Sankarapandi	38	M	166	78	Yes	No	134	82	B	8	8	8	6	Above 85	P	P	⟷	Absent	Absent	Absent	28.3	23.3	30	33.3
25	Murugan	45	M	174	72	No	No	130	80	B	23	23	8	6	Above 85	P	P	⟷	Present	Present	Present	31.6	30	41.6	35
26	Jayachandran	37	M	172	68	No	No	130	70	B	10	10	8	6	Above 85	P	P	⟷	Present	Absent	Present	21.6	18.3	25	25
27	Sivamani	31	M	162	59	No	No	130	80	B	10	10	10	6	Above 85	P	P	⟷	Present	Present	Present	25	21.6	31.6	31.6
28	Sivanesan	34	M	148	54	No	No	130	70	B	11	11	10	6	Above 85	P	P	⟷	Present	Absent	Present	21.6	18.3	21.6	28.3
29	Pandi	37	M	172	61	Yes	No	140	86	B	8	8	8	6	Above 85	P	P	⟷	Absent	Absent	Absent	18.3	18.3	23.3	30
30	Senthilkumar	37	M	171	67	No	No	110	70	B	13	13	8	6	Above 85	P	P	⟷	Absent	Absent	Absent	18.3	18.3	26.6	36.6
31	Kannan	40	M	173	76	Yes	Yes	140	90	B	20	20	8	5	Above 85	P	P	⟷	Present	Present	Present	40	43.3	31.6	35
32	Pandi	48	M	170	63	No	Yes	110	80	B	23	23	8	5	Above 85	P	P	⟷	Present	Present	Absent	21.6	28.3	36.6	40
33	Arunxavier	49	M	165	74	No	Yes	120	80	B	25	25	8	5	Above 85	P	P	⟷	Present	Present	Present	40	30	40	43.3
34	Rajendran	45	M	168	66	No	No	130	84	B	20	20	10	4	Above 85	P	P	⟷	Present	Present	Present	26.6	30	33.3	48.3
35	Suresh	40	M	159	63	No	No	128	82	B	10	10	8	6	Above 85	P	P	⟷	Present	Absent	Present	30	28.3	35	33.3
36	Periasamy	43	M	155	67	No	No	134	84	B	15	15	10	4	Above 85	P	P	⟷	Present	Present	Present	30	26.6	31.6	51.6
37	Venkatachalam	42	M	164	70	No	No	140	86	B	20	20	10	5	Above 85	P	P	⟷	Present	Present	Present	25	28.3	40	48.6
38	Selvaraj	35	M	158	68	No	Yes	130	80	B	12	12	8	6	Above 85	P	P	⟷	Present	Present	Present	25	23.3	40	36.6
39	Jeyapandi	35	M	158	75	No	No	130	80	B	14	14	10	6	Above 85	P	P	⟷	Present	Present	Present	28.3	26.6	36.6	35
40	Palanivel	36	M	174	75	No	No	140	86	B	12	12	12	3	Above 85	P	P	⟷	Present	Present	Absent	28.3	26.6	30	23.3
41	Balamurugan	36	M	162	60	No	No	130	70	B	14	14	8	6	Above 85	P	P	⟷	Absent	Absent	Absent	28.3	21.6	25	20
42	Karunagaran	39	M	162	60	No	No	130	82	B	10	10	8	6	Above 85	P	P	⟷	Present	Present	Present	28.3	11.6	40	25
43	Alagendran	37	M	158	62	Yes	No	130	80	B	15	15	8	6	Above 85	P	P	⟷	Absent	Absent	Absent	21.6	21.6	35	36.6
44	Santhanam	40	M	164	72	Yes	No	140	82	B	12	12	10	6	Above 85	P	P	⟷	Present	Present	Present	40	43.3	40	36.6
45	Murugesan	39	M	158	64	No	No	140	90	B	14	14	8	6	Above 85	P	P	⟷	Absent	Absent	Absent	25	23.3	15	13.3
46	Veerasamy	44	M	166	70	No	No	128	78	B	11	11	8	5	Above 85	P	P	⟷	Present	Present	Present	16.6	18.3	30	31.6
47	Poomibalan	38	M	172	76	No	No	130	80	B	8	8	8	6	Above 85	P	P	⟷	Present	Present	Present	18.3	23.3	21.6	31.6
48	Gangaraj	43	M	161	66	Yes	No	140	90	B	16	16	12	3	Above 85	P	P	⟷	Present	Absent	Present	25	31.6	26.6	56.6
49	Krishnan	39	M	159	67	No	No	130	70	B	11	11	8	6	Above 85	P	P	⟷	Absent	Absent	Absent	15	15	25	15
50	Murugan	34	M	155	62	Yes	No	120	80	B	11	11	10	6	Above 85	P	P	⟷	Present	Present	Absent	26.6	26.6	35	20

P - POSITIVE

CONTROL GROUP

S.No	Name	Age	Sex	Height (cm)	Weight (kg)	Smoking	Alcoholic	Systolic BP	Diastolic BP	Type of Work	No.of years of working	No.of years of exposure to noise	No. of hours of exposure/day	No. of days of exposure/ week	Decibels exposed	Rinne's test Right	Rinne's test Left	Webers' Test Centralised	PTA-4kHz Notch	4 kHz Notch Right ear	4 kHz Notch Left ear	Hearing threshold (Mid frequencies) Right ear (.5,1,2 kHZ Avg)	Hearing threshold (Mid frequencies) Left ear (.5,1,2 kHZ Avg)	Hearing threshold (High frequencies) Right ear (4,6,8 kHZ Avg)	Hearing threshold (High frequencies) Left ear(4,6,8 kHZ Avg)
1	Eswaran	46	M	165	68	No	No	140	84	OW	10	10	8	6	WNL	P	P	↔	Absent	Absent	Absent	11.6	13.3	18.3	18.3
2	Sundarrajan	34	M	174	72	No	No	130	80	OW	14	14	8	5	WNL	P	P	↔	Absent	Absent	Absent	11.6	13.3	16.6	20
3	Pandiarajan	48	M	158	78	No	No	140	86	OW	18	18	8	6	WNL	P	P	↔	Present	Present	Present	31.6	28.3	38.3	35
4	Subburaj	50	M	158	54	No	No	140	82	OW	20	20	8	6	WNL	P	P	↔	Present	Present	Present	18.3	26.6	45	30
5	Muthalagu	34	M	162	64	No	No	130	80	OW	12	12	8	6	WNL	P	P	↔	Absent	Absent	Absent	21.6	21.6	31.6	25
6	Paramasivam	46	M	167	74	No	No	136	80	OW	13	13	8	6	WNL	P	P	↔	Absent	Absent	Absent	13.3	18.3	20	21.6
7	Senthilraman	39	M	165	70	No	No	134	82	OW	14	14	8	5	WNL	P	P	↔	Absent	Absent	Absent	23.3	16.6	23.3	21.6
8	Kannan	30	M	155	53	No	No	130	70	OW	8	8	8	6	WNL	P	P	↔	Absent	Absent	Absent	31.6	26.6	11.6	26.6
9	Pitchai	42	M	175	78	No	No	136	84	OW	8	8	8	6	WNL	P	P	↔	Absent	Absent	Absent	28.3	26.6	26.6	13.6
10	Manavalan	35	M	161	58	No	No	130	70	OW	8	8	8	6	WNL	P	P	↔	Absent	Absent	Absent	13.3	15	23.3	23.3
11	Babu	42	M	158	60	No	No	110	70	OW	8	8	8	6	WNL	P	P	↔	Absent	Absent	Absent	11.6	12	26.6	21.6
12	Kesavan	37	M	171	75	No	No	140	70	OW	10	10	8	5	WNL	P	P	↔	Absent	Absent	Absent	13.3	16.6	18.3	26.6
13	Sundaram	32	M	158	59	No	No	110	70	OW	8	8	8	6	WNL	P	P	↔	Absent	Absent	Absent	12	11.6	23.3	11.6
14	Karuppannan	36	M	154	55	Yes	No	120	74	OW	9	9	8	5	WNL	P	P	↔	Absent	Absent	Absent	11.6	12	30	13.3
15	Karthik	34	M	153	54	No	No	130	72	OW	9	9	8	6	WNL	P	P	↔	Absent	Absent	Absent	13.3	13.3	16.6	11.6
16	Velan	30	M	163	61	No	No	124	72	OW	11	11	8	5	WNL	P	P	↔	Absent	Absent	Absent	13.3	13.3	11.6	18.3
17	Kumar	33	M	159	61	Yes	No	112	70	OW	8	8	8	6	WNL	P	P	↔	Present	Absent	Present	11.6	26.6	16.6	31.6
18	Karuppusamy	39	M	150	54	No	No	110	70	OW	8	8	8	6	WNL	P	P	↔	Absent	Absent	Absent	26.6	25	13.3	31.6
19	Mariappan	43	M	162	65	Yes	No	136	82	OW	11	11	8	5	WNL	P	P	↔	Absent	Absent	Absent	11.6	13.3	13.3	16.6
20	Lakshmikanthan	45	M	157	61	No	Yes	140	86	OW	16	16	8	6	WNL	P	P	↔	Present	Absent	Present	20	18.3	26.6	30
21	Alagarsamy	35	M	161	62	No	No	120	80	OW	8	8	8	6	WNL	P	P	↔	Absent	Absent	Absent	11.6	11.6	18.3	18.3
22	Sekar	40	M	149	50	Yes	Yes	134	84	OW	8	8	8	5	WNL	P	P	↔	Absent	Absent	Absent	23.3	28.6	26.6	30

23	Muthu	37	M	153	49	No	No	110	70	OW	8	8	8	6	WNL	P	P	↔	Absent	Absent	Absent	11.6	11.6	20	21.6
24	Pitchaipandi	41	M	157	61	No	No	140	80	OW	8	8	8	6	WNL	P	P	↔	Absent	Absent	Absent	48.3	40.3	26.6	26.6
25	Balakrishnan	34	M	152	57	No	No	120	70	OW	8	8	8	6	WNL	P	P	↔	Absent	Absent	Absent	30	28.3	13.3	30
26	Kanagaraj	42	M	162	68	No	No	140	80	OW	10	10	8	6	WNL	P	P	↔	Absent	Absent	Absent	11.6	13.3	13.3	11.6
27	Ranganathan	45	M	158	65	No	No	130	80	OW	14	14	8	6	WNL	P	P	↔	Absent	Absent	Absent	11.6	13.3	18.3	13.3
28	Srinivasan	47	M	165	67	Yes	No	140	80	OW	8	8	8	5	WNL	P	P	↔	Absent	Absent	Absent	18.3	11.6	11.6	13.3
29	Ravi	39	M	171	68	No	No	130	80	OW	10	10	8	5	WNL	P	P	↔	Absent	Absent	Absent	13.3	18.3	13.3	11.6
30	Suresh	35	M	169	66	No	No	120	80	OW	12	12	8	6	WNL	P	P	↔	Absent	Absent	Absent	11.6	13.3	11.6	11.6
31	Rajendran	46	M	155	65	Yes	No	150	90	OW	13	13	8	5	WNL	P	P	↔	Absent	Absent	Absent	13.3	15	21.6	21.6
32	Muniasamy	39	M	162	68	No	No	120	80	OW	14	14	8	5	WNL	P	P	↔	Absent	Absent	Absent	21.6	21.6	21.6	18.3
33	Sivakumar	43	M	140	35	No	No	140	86	OW	8	8	8	6	WNL	P	P	↔	Absent	Absent	Absent	23.3	16.6	16.6	21.6
34	Sengotayan	40	M	159	48	No	No	140	80	OW	13	13	8	5	WNL	P	P	↔	Absent	Absent	Absent	13.3	18.3	23.3	18.3
35	Kandasamy	39	M	163	66	No	No	130	82	OW	8	8	8	6	WNL	P	P	↔	Absent	Absent	Absent	16.6	18.3	13.3	11.6
36	Sakthivel	41	M	168	67	No	No	130	82	OW	8	8	8	6	WNL	P	P	↔	Absent	Absent	Absent	18.3	18.3	13.3	13.3
37	Kaveri	43	M	159	65	Yes	No	130	80	OW	8	8	8	5	WNL	P	P	↔	Absent	Absent	Absent	11.6	11.6	18.3	20
38	Arumugam	47	M	155	48	No	No	120	70	OW	13	13	8	5	WNL	P	P	↔	Absent	Absent	Absent	11.6	11.6	13.3	18.3
39	Shanmugam	37	M	163	67	No	No	140	80	OW	12	12	8	6	WNL	P	P	↔	Absent	Absent	Absent	13.3	13.3	16.6	18.3
40	Sivakumar	42	M	165	64	No	No	130	82	OW	12	12	8	6	WNL	P	P	↔	Absent	Absent	Absent	21.6	21.6	20	21.6
41	Karthikeyan	39	M	172	68	No	No	140	80	OW	11	11	8	6	WNL	P	P	↔	Absent	Absent	Absent	21.6	23.3	21.6	21.6
42	Ganesan	44	M	168	65	Yes	No	130	84	OW	8	8	8	6	WNL	P	P	↔	Absent	Absent	Absent	21.6	18.3	21.6	18.3
43	Rathnavel	38	M	161	66	No	No	130	84	OW	10	10	8	5	WNL	P	P	↔	Absent	Absent	Absent	23.3	13.3	11.6	13.3
44	Manickam	37	M	167	70	No	No	134	82	OW	11	11	8	6	WNL	P	P	↔	Absent	Absent	Absent	15	18.3	21.6	18.3
45	Govindarajan	45	M	171	64	No	No	130	86	OW	16	16	8	6	WNL	P	P	↔	Absent	Absent	Absent	21.6	20	18.3	13.3
46	Saravanan	47	M	168	59	Yes	No	130	84	OW	8	8	8	6	WNL	P	P	↔	Absent	Absent	Absent	18.3	18.3	13.3	21.6
47	Amalraj	38	M	156	58	No	No	130	80	OW	11	11	8	6	WNL	P	P	↔	Absent	Absent	Absent	11.6	13.3	16.6	13.3
48	Alagesan	35	M	169	71	No	No	140	76	OW	10	10	8	5	WNL	P	P	↔	Absent	Absent	Absent	21.6	18.3	15	18.3
49	Balu	41	M	173	68	No	No	130	80	OW	8	8	8	6	WNL	P	P	↔	Absent	Absent	Absent	23.3	21.6	21.6	20
50	Thangadurai	43	M	155	50	Yes	No	120	70	OW	8	8	8	6	WNL	P	P	↔	Absent	Absent	Absent	21.6	18.3	18.3	20

P - POSITIVE OW - OFFICE WORKER WNL - WITHIN NORMAL LIMIT